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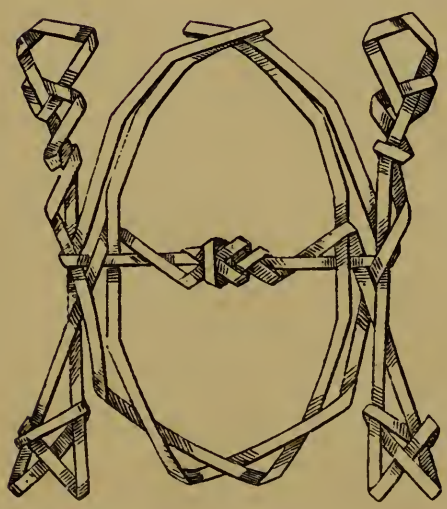

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# Ophthalmic Hospital Reports.

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Part I.

## Original Contributions.

### ON SOME POINTS IN CONNEXION WITH THE TREATMENT OF SYMPATHETIC OPHTHALMIA,

By George Lawson, F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital.*

The causes of sympathetic ophthalmia are now fairly well recognised; but how to avert the disease, and how to treat it when established, are still open questions.

Before considering these two points it may be well to recall the peculiar characters of the disease.

They may be summarised thus:—

1. The disease is induced in one eye by irritation set up in the other, generally either as the result of injury, or from degenerative changes going on in a damaged or lost eye.

The disease usually commences *without* pain as a warning, though it is commonly preceded by a certain amount of irritation, slight photophobia, impairment of accommodation sufficient to give rise to fatigue of the eye, and lacrimation.

One or all of these symptoms may be present, and in an adult would probably draw attention to the state of the eye, but in a child they are not, as a rule, sufficient to lead him to make complaint, so that in children the disease is not unfrequently far advanced before it is detected.

2. The inflammation is fibrinous or plastic, and, I believe, starts in the ciliary region, and extends simultaneously to the iris and choroid. I have never seen a sympathetically inflamed eye suppurate.

3. After a variable period the tension of the globe increases, and in this stage there is pain, which is sometimes very severe. If the disease be very acute, ciliary staphyloma will occur at this stage. Increased tension may continue for many months, and when staphyloma has commenced it will go on increasing. In the majority of cases, however, the staphyloma does not occur, and after a variable time the excessive tension subsides, then becomes normal, and ultimately the eye softens. The eye is then irreparably lost.

I. *How to avert the Disease.*—My own experience leads me to believe that if an eye have received a severe injury, and it be excised before any symptoms of irritation occur in the opposite eye, then the sound eye is safe from sympathetic ophthalmia.

There may be a few apparent exceptions to this rule, but it has not yet been my misfortune to meet them. I have seen cases in which there has been very slight irritation only in the uninjured eye, and for this the injured eye has been removed. After the operation the irritation has completely subsided, and the safety of the eye has apparently been secured. But after a lapse of time varying from a few weeks or months to many years, the irritation has recurred, and rapidly drifted into the true sympathetic irido-choroiditis, ending in total loss of the eye. These cases can, however, hardly be considered as exceptions to the general rule which I have laid down. Sympathetic irritation is closely allied to sympathetic ophthalmia, and although it presents features different from true sympathetic inflammation, yet it may and frequently does drift into it, as rodent ulcer, which in its early stages is distinct from epithelioma, may at a later date pass into and assume all the characters of this formidable disease.

Now one of the peculiarities of sympathetic ophthalmia is

that it is recurrent, as indeed are all the forms of iritis, no matter how induced; and sympathetic irritation shares also this characteristic, that it is recurrent, and an eye which has once been subjected to sympathetic irritation may at some future period have a recurrence which may drift into well marked sympathetic ophthalmia. Such I think is the explanation of the two following cases :—

CASE I.—*Sympathetic Irritation coming on Seven Months after an Injury—Injured Eye removed—Complete Recovery and Good Sight for nearly Nine Years—Recurrence of Inflammation—Loss of Sight—Staphyloma—Sclerotomy.*

Robert W., æt. 33, was admitted into the Royal London Ophthalmic Hospital on June 6, 1880. Ten years before, he received an injury to the right eye from a blow with a small piece of wood, which from his description appeared to have ruptured the globe. Seven months after the injury he had sympathetic irritation in the left eye, and for this the right eye was excised. All irritation ceased after the operation, and the recovery was complete. He was sure that he could see with the eye "as well as he ever did in his life." He could see the smallest type, or the markings on the minute-hand dial of a watch. For nearly nine years he continued quite well. About twelve months before admission he had slight pain in the eye with failure of sight, and this increased until he applied at Moorfields Hospital, when he was admitted as an in-patient.

On admission the eye presented all the well marked appearances of advanced sympathetic ophthalmia. There was complete posterior synechia; the pupillary space was filled with opaque membrane; the iris, and especially its peripheral portion, was bulged towards the cornea, so that the anterior chamber was almost obliterated. There was a general bulging of the ciliary region, with a thinning of the sclerotic, and at the upper and inner part there was a large irregular staphylomatous protrusion. The tension of the eye was but a little above normal. The man could not count fingers, and had only perception of light in the outer part of the field. He had severe constant pain in the eyeball, which kept him awake at night.

In this patient the disease may be said to have run its course. His sight was reduced to a mere perception and he had staphyloma; but in addition he had pain, and it was to the relief of the pain that the treatment was now directed.

On June 18 sclerotomy was performed. A thin Graefe's cataract knife was made to puncture the sclerotic in the upper region of the eyeball, close to the corneo-sclerotic junction, and carried across the anterior chamber in front of the iris, the counter-puncture being made at a corresponding point on the opposite side, and the knife withdrawn. So shallow was the anterior chamber that a few of the fibres of the iris were caught by the point of the knife as it traversed it.

The result of this operation was very satisfactory. On June 29 the staphyloma had quite subsided, and the eye was quite free from pain. The sight was not materially improved. The eye was soft, —T. 1.

Case II.—*Sympathetic Ophthalmia coming on Four Weeks after an Injury to the Opposite Eye—Excision—Complete Recovery—Recurrence of Inflammation—Loss of Sight.*

Fred. H., æt. 9, was admitted into the Royal London Ophthalmic Hospital on the 23rd of January, 1880, four days after receiving the following injury. Whilst playing, he was struck on the left eye with a piece of stick which was thrown at him. On admission there was a small vertical rent on the inner side, but within the margin of the cornea, through which the iris was prolapsed, and there was traumatic cataract. The prolapsed iris was snipped off. Between the fourth and fifth week symptoms of irritation came on in the right eye, and the injured eye was at once excised. After the operation all the symptoms of irritation subsided, and the eye apparently completely recovered. The child could see the smallest type, and had good distant vision.

On April 2 the lad was brought to the Hospital complaining of a slight recurrence of irritation in the eye. There was some lacrimation and a little ciliary injection. These symptoms increased in spite of treatment.

On May 7 the boy was admitted into the Hospital, where he

continued until the end of June. There was at this time complete posterior synechia, lymph in the pupillary space, and a very shallow anterior chamber. The eye was quiet, and could count fingers at six feet, and read letters of Jaeger 16 and 18.

II. *How to proceed—(a) when the Injured Eye is totally Blind; (b) when the Injured Eye still retains some Sight.*—An eye has received an injury, and sympathetic ophthalmia has occurred in the other eye: Should the injured eye be removed? Experience has convinced me that the removal of the injured eye when sympathetic ophthalmia is established in the other eye will not arrest the disease; the inflammation will go on as if the source of the primary irritation remained. Nevertheless, if the injured eye be hopelessly blind, it should, I think, be excised, in order to give the patient the chance of any benefit that might accrue therefrom.

The removal of the injured eye may arrest sympathetic irritation, and frequently does, but it will not stay the progress of sympathetic ophthalmia. It is, therefore, doubtful whether any good will ensue from the removal of the injured eye. On several occasions, where the injured eye has retained some sight, I have seen the opposite eye destroyed by sympathetic ophthalmia, while the injured eye has ultimately so recovered that useful sight has been restored, and the patient has been able to get about without assistance. The conclusion at which I have arrived from watching these cases is, therefore, that if sympathetic ophthalmia be established, the injured eye should not be removed if it retains any sight.

III. *How to treat the Disease.*—In the curative treatment of sympathetic ophthalmia we have much to learn. At present we can do but little, as we have no means by which we can control the disease with certainty. The mode of treatment which seems to benefit in one case fails to afford any relief in another. On one point I feel quite satisfied: that operative interference in the inflamed eye is positively prejudicial in the early stages of sympathetic ophthalmia. I feel also equally certain that in the later stages when the pupil is

closed and false membrane fills the pupillary space, that no operation should be done except when the eye is absolutely quiet, and there is no inflammatory action.

My present feeling is that *in the early stage* of the disease the eye should be excluded from all light. The darkest possible glasses should be worn out-of-doors, and whilst at home the patient should be in a dark room with a black bandage across the eyes, provided the bandage does not produce discomfort by occasioning heat and conjunctival secretion from the lids. The aim is to give the eye as perfect rest as possible. During this stage mercurial inunction into the temple every night may be tried and continued for two or three months, care being taken to avoid salivation by diminishing or omitting the ointment for a few days as required. At the same time quinine in from half grain to two grain doses, according to the age of the patient, combined with small doses of the extract of belladonna, may be given two or three times a day. A weak solution (0·25 per cent.) of atropine or daturine may be dropped once a day into the eye, but in the later stage of the disease, when the whole posterior surface of the iris is adherent to the capsule of the lens, I believe that all mydriatics do harm by stimulating the dilating fibres of the iris to contract when from the adhesion of the iris to the lens-capsule they cannot act. Mydriatics also favour the development of the state of increased tension which usually comes on at a later period of the disease.

In *the second stage* of the disease when the union between the iris and lens-capsule has been effected, but after the acute symptoms have subsided and the eye is quiet, it is a question whether an operation may be attempted to improve the sight. My opinion is that if the sight is sufficient for immediate requirements, and will enable the patient to walk about without assistance, the eye should be left alone. It is the only eye, and operations on eyes sympathetically inflamed are so unfavourable that it is better to let the patient enjoy the sight he has, rather than to risk the loss of it with the prospect only of a slight improvement.

If, however, the sight is so defective as to be almost useless, and there is a fair field of vision, then an attempt should be made to improve it by an operation. I have for a long time felt satisfied that the great object to be attained in these cases is to get rid of the lens, as I have noticed that when this has been satisfactorily accomplished the tendency to the recurrences of inflammation is greatly diminished, and an artificial pupil may be afterwards made, through which the patient will enjoy greatly improved sight. The removal of the lens is an operation of difficulty. It may be accomplished thus:—First make a section of the cornea with a Graefe's cataract knife or a broad keratome, then tear open the pupil and through the capsule of lens with a cystotome, or if this be insufficient, remove a portion of iris and the adherent lens-capsule with a pair of iris scissors, and, lastly, with a little pressure on the globe with the back of the curette cause the escape of the lens through the corneal wound. This operation may generally be accomplished without the loss of any vitreous, and the eye will usually recover from its effects well, but the pupil will probably again become closed. Another operation will afterwards be required for the formation of a new pupil.

*The third stage* of the disease is characterised by increased tension of the globe, and this condition is generally associated with pain, oftentimes very severe, and sufficient to make the patient willing to submit to any means suggested for his relief. This state of tension lasts a variable period, but, as a rule, it ultimately subsides, and the eye softens. If the state of tension be long continued the little sight that is left soon vanishes. When the eye has reached this stage of increased tension active treatment is necessary. An attempt to do an iridectomy I have always found unsuccessful. The iris is adherent throughout its posterior surface to the lens-capsule, and is either so rotten that the forceps pass through it, and cannot seize a piece to draw it out, or else the iris has become tough and fibrous, and simply tears like calico under the grip of the forceps, and no portion of it can be satisfactorily removed.

During the last eight months I have performed the operation of sclerotomy in these cases, and have been pleased with the result. The knife has been made to pierce the sclerotic just external to the corneo-sclerotic junction, rather higher than the point selected for a Graefe's extraction of cataract, and carried in front of the iris; the counter-puncture has been made at a corresponding point on the opposite side. After slight cutting, so as to make the section a little broader than the breadth of the blade, the knife has been withdrawn, leaving a wide bridge between the sections.

In each instance the pain has been relieved immediately, and in two (Cases I and III), in which there were ciliary staphylomata, the bulgings subsided, and the eyes became soft. In one patient (Case III), in whom the pain was so excessive, that morphia had to be injected subcutaneously twice a day, the relief was so rapid that after the operation the patient slept without the morphia injections, which were not again required. In this case the sight also was improved. Before the operation the man could only just count fingers when held close to the face, and when he left the hospital he could read Jaeger 19 at 2 feet.

Case III.—*Sympathetic Ophthalmia coming on about ten weeks after the Injury—Loss of Sight—Increased Tension—Ciliary Staphyloma—Sclerotomy—Good result.*

Edward T., a porter, æt. 31, was first admitted into the Ophthalmic Hospital on August 8, 1879. His right eye had been injured by the explosion of a soda-water bottle in the previous May, and the sight of the left eye began to fail about seven weeks afterwards.

On admission there was a puckered cicatrix in the ciliary region of the right eye, and the globe was shrunken. In the left eye there was a slight ciliary blush. The pupil was very sluggish, almost inactive, and a thin film of lymph occupied the pupillary space. There were minute dots of lymph on the posterior surface of the lower half of cornea. This eye could only read Jaeger 16. Tension was normal.

The injured eye was excised. The patient remained under

treatment until September 22, when he was discharged. The eye was quiet, and had so much improved that he could read Jaeger 2 at 10″ and V = $\frac{20}{40}$.

On November 7, 1879, the patient was readmitted into the Hospital with a recurrence of the sympathetic inflammation of the left eye. There was complete posterior synechia, and the pupillary space was filled with opaque membrane.

The vision was very bad. He could hardly see fingers. He was discharged on the 29th. The eye much quieter and less injected. Sight not improved.

On December 12, 1879, the patient was again readmitted. Soon after returning home he had had another attack of inflammation of the eye, accompanied with pain, which was relieved by leeches and blisters to the temple. The pupil was on readmission excluded and occluded, and the iris was bulging. T. 1.

On December 28 he had great pain; a sense of burning in the forehead and eyeball; much ciliary injection, lacrimation, and photophobia. T. 2. Ordered two leeches to temple.

From this time the symptoms increased in severity. The pain became so severe that relief could only be obtained by subcutaneous injections of morphia, given always once, and frequently twice a day.

Other sedatives were tried, but none gave the relief of the hypodermic injections of morphia.

The tension of the globe continued T. 2. Drops of sulphate of eserine were tried, but without influencing the tension. The sclerotic in the upper and outer half of the ciliary region began to thin, and slightly bulge. The sight was so reduced that he could only count fingers held very close to the face.

February 13 the patient was placed under ether, and a sclerotomy was made upwards, a good bridge of sclerotic being left. The relief from the operation was immediate. The patient passed a good night without the hypodermic injection of morphia, and next morning the tension was normal.

On the 24th the iris was still bulging, but there was no pain. The patient could read Jaeger 19 at 2 feet.

On March 3 he was discharged. The tension of the globe had slightly increased. He was free from pain, and he could read letters of Jaeger 16.

## ON A THEORY OF GLAUCOMA.

By W. A. Brailey, M.D.

By the term glaucoma, I mean not only primary glaucoma inclusive of its hæmorrhagic form, but also the cases where increased tension is secondary to injury, corneal ulcer, tumours, or to various inflammations of the uveal tract.

The eye is a capsule of fluid, with walls extensible in youth, but becoming more rigid as age advances. The fluid in it, like the tissues of the body generally, is in a state of constant change. Its ingress probably varies somewhat in health with the conditions of the circulation. The channels of egress are doubtless sufficient for amounts varying within considerable limits.

But, when compared with other parts of the body, for example, areolar tissue, the capacity of the eye for accommodating an excess of fluid is limited. Moreover, the tissues liable to compression here are both delicate and important.

An accumulation of fluid, whether due to an inflow beyond what can be carried off by the normal channels of outflow, or to obstructed outflow, or to both causes acting together, constitutes the condition of increased tension called glaucoma.

The theory of an increased inflow has had Donders as its distinguished exponent, by whom it was attributed to a neurosis.

That of obstructed outflow has been maintained by Knies, Weber, Wecker, and others. These agree generally in putting the principal seat of obstruction at Schlemm's canal, whether by its inflammation or by the apposition of the iris to its entrance. Their differences mostly concern the way in which this apposition has been effected; some attributing it to the swelling of the ciliary folds, others to the advance or an alteration in shape of the lens. In the opinion

of the author both principal theories have their value, and each is capable even by itself of accounting for tension.

He attributes increased inflow sometimes to a visibly increased blood supply, and sometimes to inflammation, especially of the ciliary body and iris. Diminished outflow he ascribes, in most cases, to an obstruction in the region of the ligamentum pectinatum, and in some few, in the exits from the perichoroidal space. He finds, however, in most cases such a condition of the optic nerve as would hinder any passage of fluids along it; though that this is a channel of exit in the normal eye is unproved by experiment. Obstruction at the entrance to Schlemm's canal, he ascribes in most cases to valvular action of the iris, induced either by its position or by the condition of it, and the ciliary body.

The fluids in the eye may be divided for convenience into three systems:—

A. That of the vitreous and aqueous chambers, inclusive of any found in the subhyaloid space.
B. That of the retina.
C. That of the choroid.

The two last of these are constantly recruiting the fluids of the first, and any accumulation in them is rare. On this account they have not often much direct influence on the tension. They will be described hereafter, and their influence discussed.

The fluids contained in the meshes of the vitreous and in the aqueous chambers are in pretty free communication across the so-called canal of Petit external to the lens margin, and through the pupillary aperture. They are recruited by the transudation which has taken place from the blood-vessels for the nourishment of the tissues, more especially from those of the uveal tract, as containing by far the greatest bulk of blood within the eye. And of this tract the iris and ciliary body are, perhaps, the most important parts, as being immediately in contact with the vitreous; whereas the choroid is separated from it by the retina, which must offer at least some obstruc-

tion to the passage of fluid through it. Some conditions of detachment of retina appear to point to this: and some conditions of cysts of the iris afford indications that considerable fluid escapes from its uveal surface. Fluid from the proper retinal blood-vessels without doubt drains into the vitreous cavity, but its amount is comparatively small, as they are far less in size and number than those of the uveal tract.

In the body generally it has been shown that the amount of transudation varies with the blood-pressure. This pressure is increased locally whenever any artery is dilated up to its origin from a larger trunk. The pressure in its capillaries becomes also increased beyond the normal, and an increased escape of fluid results.

This enlargement of the vessels is seen *within* the eye in numerous cases (see Table, page 94). It affects almost solely the blood-vessels of the ciliary region and iris. *Outside* the eye the vessels forming part of the supply of this region, the anterior ciliary arteries, are seen during life enlarged in many cases in their course beneath the conjunctiva.

This enlargement is probably the consequence of an affection of the vaso-motor nerves, and a *neurosis* theory will explain the occasional association of glaucoma with mental anxiety and with neuralgia. Indeed, it has been established by experiment that the 5th and sympathetic nerves have an influence on the tension. The speedy onset and occasional spontaneous subsidence of glaucoma will also be thus explained.

The arterial dilatation may be independent of visible external causes, or it may arise in consequence of a blow or wound, and constitute by itself the cause of a glaucoma (see Group III, page 114). Its existence is by no means necessary as a cause of tension, though it is found in many cases of primary, and still more frequently in hæmorrhagic glaucoma. I think its existence may be due in some of these cases to irritation of the nerves of the ciliary body consequent on the extreme stretching of the fibres of the suspensory apparatus of the lens. The mode in which this stretching

is set up in consequence of atrophy and alteration of shape of the ciliary body will be subsequently explained. The same explanation will apply to the occasional extremely rapid occurrence of tension after the lens has been partially or entirely displaced backwards by violence, and sometimes also to cases where glaucoma has quickly followed the forcible breaking up of cataracts by needling.

I infer generally that arterial enlargement if not found at excision has never been in existence. It is possible, however, that if it have not been of so long duration that the usually occurring atrophy of the arterial walls have not supervened, it may have existed, and then afterwards subsided. Such a suggestion would explain the phenomena of intermittent glaucoma, and the occasional cure of glaucoma by paracentesis alone.

An inflammation of the ciliary body and iris of a kind ultimately passing into atrophy or into atrophy with sclerosis, has been described in another paper* as frequently existing especially in young subjects with disease of short duration. The opinion has been expressed there, that some inflammation always precedes the atrophy, that it exists prior to the establishment of the tension, and that in most cases of Primary Glaucoma, especially in old persons, its degree is but slight.

In such case it will have little effect in increasing the fluids of the aqueous and vitreous chambers, and any excess derived from it will be easily carried off by the natural channels. It will probably be different if the inflammation be acute, and here we may presume a hypersecretion which will constitute a material addition.

But when sclerosis and atrophy are established, there will be rather an actual diminution of the fluids normally recruiting the vitreous and aqueous, unless an enlarged arterial supply exist.

It has been experimentally established that the excess fluids of the vitreous and aqueous chambers have their normal exits from the anterior chamber through the endothe-

* "On the Condition of the Optic Nerve, Ciliary Body, and Iris, in Increased Tension," page 86.

lium-covered meshwork of the ligamentum pectinatum, after passing from the vitreous chamber, where they are probably derived from the choroidal and retinal vessels, through the canal of Petit, where they are recruited from the ciliary folds, into the posterior chamber of the aqueous humour, where is added to them the fluid escaped from the uveal pigment stratum of the iris, and through the pupillary aperture.

In consequence of this current, which is certainly very slow in health, there is normally some slight excess of pressure behind the iris over that in the anterior chamber, though this power is by no means sufficient to move the thick and contractile diaphragm formed by the normal iris.

It has, however, been established that a very slight increase of pressure in the vitreous over that in the aqueous chamber, is sufficient to carry forwards in the eyes of recently killed pigs the periphery of the iris, and block further exit through the ligamentum pectinatum.*

If such a movement ever occur in living human eyes, it is probably of extremely rare occurrence with unaltered ciliary body and iris.† For example, it has not taken place even in Case 352, nor in 101 of Group III. Some cases, however, seem to show that it may be induced without any increased current of fluid, provided that the periphery of the anterior chamber has been so narrowed in an antero-posterior direction as to cause a difficulty in the passage of fluid between the iris and cornea towards the ligamentum pectinatum. For then the pressure on the front surface of the peripheral part of the iris, will tend to become decidedly less than that on its posterior surface. This condition of things is brought about by the atrophy of the ciliary body as elsewhere explained (page 86). Under these circumstances, it is quite intelligible that a very slight additional atrophy at any one point will accentuate the difference of pressure and carry forwards the iris there. The pressure on the front surface of

* Priestley Smith "On Glaucoma," p. 133.

† It is obvious that during life the conditions of the experiments just referred to are only fulfilled when the cornea is perforated.

the immediately adjacent parts of the iris will then also be diminished, and thus the application of the iris periphery will spread laterally till its entire circuit is involved, or at least as much of it as is implicated in the atrophy of the ciliary body.*

There are, however, other cases with applied iris in which the atrophy of the ciliary body, though present in a certain degree, obviously could not have advanced the periphery of the latter so much as to have induced its valvular action without increased pressure behind it.

For in these the iris is applied at a point distinctly in advance of its base, which may even be separated from the front wall of the extreme periphery of the chamber by a little fluid imprisoned there (see page 91). This condition of things can only happen when the chamber is extended backwards by the atrophy and alteration in shape of the ciliary body.

I attribute the position of the iris in these cases to an increased flow of fluid towards Schlemm's canal from the ciliary body, due either to its inflammation or to the increased vascular supply already referred to. The valvular action of the iris is further facilitated by its atrophy, which is always developed in connexion with the inflammation or atrophy of the ciliary body, and by any shallowness of the anterior chamber. The disposition to it is increased if there have been anterior synechia, or if the eye be hypermetropic.

It is perfectly clear that in cases of perforated corneal ulcer the iris acts as a valve. For whether, as is far more usual, it line the entire posterior corneal surface, or be applied only partially as explained in another paper, its effect is to prevent exit of the fluid from behind it.

Its application is due to the sudden diminution of pressure on its anterior surface, and so long as any leakage continues through the region of the ulcer, the anterior chamber cannot be re-established. If, however, the perforation be so limited that it can be completely plugged by the iris and

* The Ligamentum Pectinatum is by its shrinking the especial cause of the approximation of the iris periphery to the cornea (see pages 92, 93).

thus rapidly closed, the anterior chamber may be retained, and the eye be left with only a point-like anterior synechia. If the perforation be large, the iris, if it has not, when the leakage ceases, attained to the atrophic condition to which it is rapidly progressing, may, by the secretion of fluid from its anterior surface, partly re-establish the anterior chamber.

The failure of this secretion in advanced cases of glaucoma is the reason why the lens and iris sometimes remain jammed up against the cornea after operations, giving rise to the condition known as Malignant Glaucoma.

If the entrance to the canal of Schlemm be blocked by inflammation, as appears to be the case in "Iritis Serosa," the pressure on the two sides of the iris will be equal, and it will have no tendency to advance even though the tension be extreme.

It is obvious that exclusion or occlusion of the pupil will also prevent escape of fluids from behind the iris through the canal of Schlemm and thus cause glaucoma.

A rapid increase of the contents of the eye, as in suppurative panophthalmitis, will also cause increased tension, while one slowly progressing as in the case of many tumours, will be compensated for by atrophy of neighbouring parts, the vitreous for example, unless a channel of exit be obstructed by its growth.

With regard to the fluids of the retina, their source and mode of exit are more matters of speculation, being rather inferred from pathological conditions than determined directly by experiment.

The positions in which leucocytes are found in some diseases (see page 138), appear to show that fluid from the proper retinal blood-vessels drains through the internal retinal surface. Some may also find its way along the sheaths of the retinal vessels into those of the central vessels of the nerve.

The disease of the retinal vessels which exists in all cases of Hæmorrhagic Glaucoma, and also in most other cases of Primary Glaucoma, is fully described later (see page 133).

It has no bearing on the tension, for a similar condition of the vascular walls without tension is found in the eyes as well as in other organs of many patients who are the subjects of various general diseases.

Moreover, there is evidence that in Glaucoma it may either exist previous to or be developed subsequent to the tension (see Case 483).

The fluid of the outer retinal layers is undoubtedly derived from the chorio-capillaris. It probably finds its principal exit towards the vitreous cavity, thus completely traversing the retina in its course. Some, however, appears to escape by means of the optic nerve, either along the sheaths of the central vessels in company with that from the internal retinal layers as previously described, or along the lymph-spaces of the fibrous stroma of the nerve. The function of the optic nerve as a channel of exit is, however, as yet unproved, but if existing it would be interfered with by the sclerosis of the lamina cribrosa and nerve elsewhere described as existing in Glaucoma (see page 87).

There are, sometimes, in this disease indications of an accumulation in the inter-granule layer near the optic disc, and more often in the neighbourhood of the ora serrata. Besides this, there are very often similar indications of an accumulation in front of the lamina cribrosa, and there is, sometimes, traceable a fissure or layer of looser tissue leading to this from the inter-granule layer. Where the retina is detached the stratum of loose œdematous tissue appears to communicate with the sub-retinal space.

These appearances would be consistent with the existence of a current from the outer retinal layers to the lymph channels of the optic nerve.

In Glaucoma the retina is sometimes found extremely cystic, corresponding to where the sclerotic is staphylomatous over a small area of inflamed and thinned choroid. The explanation of this is not so clear, though the same ideas as to the escape of fluid from the chorio-capillaris might be applicable here.

The relative quantity and effects of the fluids derived from the retinal blood-vessels must in any case be small. Moreover, there are never indications that the retinal blood-supply is increased in Primary Glaucoma, while it is certainly diminished when the tension has been fully established.

And if sclerosis of the nerve and lamina cribrosa be a means of retaining some of the waste fluids of the retina within the globe, it has, when unaided, no effect on the tension, for it exists together with normal tension in many cases of cerebral disease.

The Choroid derives its fluid from its own blood-vessels, and has probably one of its outflows into the retina as above stated.

But some fluid, especially from its outer layers, escapes by way of the perichoroidal lymph space into the cavity of the capsule of Tenon along the spaces surrounding the venæ vorticosæ and the long ciliary arteries and nerves.

There is no reason to suppose this outflow interfered with in any of the cases given in the Table (p. 94), but there is evidence of increased fluid in the perichoroidal space in some few (see Nos. 214 and 274).

This fluid would be increased not only from inflammation of the choroid attended by hypersecretion as in choroiditis serosa, but from obstructed efflux through tumours involving or pressing on the outlets.

It is difficult to judge of the effects of choroiditis serosa alone, as it rarely, if ever, occurs without iritis and cyclitis, the secretion from which has its exit by the canal of Schlemm.

It will be seen that none of the cases given in the Table are without one or more of the conditions of diminished efflux or increased afflux to which we have attributed increased tension.

Out of the entire number of cases reported in the Table, two have sclerosis of optic nerve only. (It is coupled, however, in each case with disseminated choroiditis.)

Forty have access to the canal of Schlemm obstructed, and inflammation or sclerosis of the optic nerve.

Four have only enlarged arteries in the ciliary region, and inflammation or sclerosis of optic nerve.

Thirty-eight have enlarged arteries in the ciliary region, access to canal of Schlemm obstructed, together with inflammation or sclerosis of the optic nerve.

One has access to the canal of Schlemm obstructed by an universal posterior synechia, as well as a little enlargement of the arteries, together with inflammation and sclerosis of the optic nerve.

Three (Group VI) have iris periphery free, no atrophy of it or of ciliary body, some neuritis, also inflammation of iris, ciliary body and choroid, with evidence of serous hypersecretion.

Testing this short account of the principal agents concerned in the theory by the Table of cases given at page 94, we find that whenever the canal of Schlemm is blocked by the iris, this condition is accompanied either by perforated corneal ulcer or by an acute inflammation of the ciliary body, with little or no atrophy, or more commonly by an extreme atrophy of the same with or without sclerosis. Any of these conditions may be accompanied by enlarged arteries.

In Group I (Primary Glaucoma) we find the canal of Schlemm closed, or access to it extremely narrowed in 93 per cent. of the cases. Access to it is free in 7 per cent. The ciliary arteries are enlarged in 50 per cent. The canal of Schlemm is obstructed without arterial change in 12 out of the 29 cases.

It is thus clear that obstruction at the canal of Schlemm is sufficient to maintain a glaucomatous condition, and even to originate one, provided that the iris have been brought into the required position.

There are in this Group two cases (Nos. 406 and 265) exceptional as regards the canal of Schlemm and the iris adhesion. Nor do we even find in them sufficient inflammation of the ciliary body to warrant us in suggesting this as the cause of the tension. They will, however, both be seen to

have sclerosis of the optic nerve, and in the first there is also a distinct inflammatory affection of the choroid. Being thus qualified, and constituting such a minority of the cases in the Table, I shall not further discuss them here, but leave my readers to judge their value in relation to the theory suggested, as their principal pathological conditions are fully reported in the Table.

In Group II (Hæmorrhagic Glaucoma) access to the canal of Schlemm is obstructed in every case, and the arteries are enlarged in a greater proportion than in Group I. Thus we should expect to find the Glaucoma more acutely developed here, which is in fact the case. Moreover, the atrophy and sclerosis are also greater than in Group I. No exceptional case is found here.

Group III (Glaucoma after accidents), which by the conditions under which it is formed contains cases of short duration, with little inflammation at the seat of injury, has consequently usually but little inflammation or sclerosis of the ciliary body. Therefore, we are prepared to find the iris periphery not applied in some cases. The enlarged arteries, on the other hand, are of far more frequent occurrence than in the other groups, being present in 87 per cent.

There appears to be one partially exceptional case, No. 214, but in it the iris is applied at one part, and there is present, as in one of the exceptional cases of Group I, some disseminated choroiditis.

In Group IV (Corneal ulcers) the canal of Schlemm is blocked in every case, but sclerosis and enlarged arteries are decidedly more rare. The muscle is always somewhat inflamed at an early stage, and later tends to atrophy. If an adult or old person be the subject of the ulcer, there may also be sclerosis. The optic nerve is always affected. Neuritis with an inflammation of the tissue between the innermost and outermost nerve-sheaths, makes its appearance very speedily after the development of the corneal affection, and before the establishment of the tension. It generally passes on to sclerosis.

Group V (Buphthalmos) is the same as Group IV, insomuch that access to the canal of Schlemm is blocked in every case, and also in other respects.

In Group VI (Iritis serosa) the ligamentum pectinatum and neighbourhood of the canal of Schlemm are inflamed in every case. There is much inflammation of the ciliary body and iris, but no atrophy. The iris periphery is widely removed from the cornea, except where in cases 285 and 509, it has become partly entangled in the iridectomy wound.

Most theories of Glaucoma hitherto advanced rest on an obstructed outflow through the ligamentum pectinatum either by an inflammation or by apposition of the peripheral part of the iris. Their differences consist in the mode in which the iris is supposed to acquire this position.

Some attribute it to an advance of the lens from increased pressure from behind it.

But it appears to be almost impossible that any advance of the lens could produce this effect. For in my microscopic specimens where the lens capsule is left with its normal attachment to the ciliary body, its periphery is on a level considerably internal to the canal of Schlemm. And the extreme angle of the anterior chamber, the point where in many cases with great atrophy of the ciliary body the adhesion appears to begin, is normally even a trifle more external than Schlemm's canal.

And in the pseudo-glioma often seen in young children, while the centre of the lens is distinctly advanced very near to the cornea, the peripheral part of the anterior chamber is deepened by the drawing back of the most circumferential part of the iris.

Such a condition is also not uncommon in adults in whose vitreous chamber organised inflammatory products have advanced the lens, while retracting the iris periphery.

An extreme advance of the lens, as after paracentesis of the cornea, would prevent access to the canal of Schlemm by pressing the *pupillary* part of the lens against the cornea.

Then the iris also would apply itself and prevent further approach to the canal of Schlemm. But this certainly does not take place in the course of a Glaucoma.

It is more plausible that the ciliary folds swollen by vascular injection could have advanced the periphery of the iris. Only a few of my specimens, however, give any support to this explanation. Moreover, the swollen folds should have produced their effect also in iritis and cyclitis serosa, and in cases of Glaucoma with unusually deep anterior chamber. The theory fails also to explain the absence of advance of the iris in some cases of Group III.

Others place the obstructed exit at the canal of Petit by an increased diameter of the lens occurring with age. No measurements, as far as I am aware, are extant showing the increase of the lens upon which this theory is based. Moreover, one would *à priori* expect a shrinking in this direction coincident with the antero-posterior diminution that undoubtedly takes place.

Such an atrophy of the ciliary body as has been described, if occurring before the closure of the canal of Schlemm, would widen the space surrounding the lens margin. Moreover, this theory will not explain the occurrence of Glaucoma in young subjects, nor the frequent rapid development of tension in an eye whose lens has been dislocated into the vitreous chamber by a blow (see Cases 101 and 421) nor the occurrence of Glaucoma after cataract extraction without entanglement of the iris or occlusion of the pupil.

Every theory of Glaucoma though based on howsoever many examinations of eyes excised on account of severe disease, is open to the objection that the changes upon which it rests may have arisen subsequently to the establishment of the tension. It would require *post-mortem* examinations of very early cases in order to show that inflammation, atrophy, and sclerosis, whether of muscle or nerve, have been in existence previously. And this good fortune does not fall to the lot of everyone. Schnabel, however, a most competent observer, in referring to the author's previous paper on

Atrophy of the Ciliary Muscle, records* the pathological examination of a case in which he found the atrophy of the ciliary muscle distinctly developed with tension of only a few hours' duration.

Moreover, both atrophy and sclerosis are fully established in some cases of a few months' history only (see Group I, Cases 241 and 763).

On the other hand, some cases of injury (see Group III) prove conclusively that atrophy and sclerosis are developed with extreme rapidity.

But again, many phenomena are seen in eyes about to become glaucomatous before any increase of tension can be clinically detected. Of these, the change in refraction, the diminution in accommodative power, and the sluggish pupil would be explained by the atrophic changes in the shape and texture of the ciliary body and iris, whether the result of a slow inflammation imperceptible to the eye of the surgeon, or non-inflammatory.

The occurrence of Glaucoma without such premonitory symptoms would conform with the enlargement of arterial supply, and, perhaps, even with the establishment of an acute inflammation. I have before suggested that the inflammation of the ciliary body and iris is attended with increased fluid. It is of a kind leading rapidly to atrophy, which is even more conspicuously seen in the iris than in the ciliary body. The microscopical appearances in my specimens conform more frequently with the hypothesis of fluid pressure than with any other.

Reasons have been before given why the iris base should not, in normal conditions, act as a valve. But the case is altered when with an atrophied iris, more especially one whose base is already somewhat approximated to the cornea by ciliary atrophy, we have an increased escape of fluid from behind it.

With these three circumstances combined, it is possible to conceive the iris base advancing under pressure of the current of fluid.

* Knapp and Moos, Archives of Ophthalmology, vol. vii, p. 279.

An extremely slight inflammation of the ciliary body would not be attended with sufficient hypersecretion to account for the movement of the iris, but the atrophy subsequent to it would so approximate the iris to the cornea, that it would be under the influence of the normal or even slightly diminished flow of fluid towards Schlemm's canal.

## A FURTHER INVESTIGATION OF THE PATHOLOGY OF GLAUCOMA.

By Priestley Smith,

*Ophthalmic Surgeon to the Queen's Hospital, Birmingham.*

In an essay on glaucoma,* published in October last, I advanced a theory which was to a certain extent new. It was founded in part upon the discoveries of Leber, Knies, and Weber, in part upon the results of certain experiments of my own. It was supported by a good deal of circumstantial evidence drawn from the clinical aspects of the disease; but it was at the time of writing a theory and nothing more—proof of its truth was wanting. As the result of further investigation, I am now able to offer some additional evidence in connexion with certain of the doubtful points.

A brief re-statement of the steps by which the theory was arrived at, and of the theory itself, will be convenient here.

*a.* The intraocular fluid escapes from the interior of the eye, at the angle of the anterior chamber (Leber).

*b.* In glaucomatous eyes microscopically examined, this angle is found to be narrowed or closed. In the great majority of cases a firm adhesion exists between the periphery of the iris and the opposing surface of the cornea (Knies, Weber).

*c.* It is manifest that this closure is connected with the undue retention of the intraocular fluid, and the excess of tension, which characterise glaucoma, especially when we remember that the only operative procedures which have a definitely curative action on glaucoma are practised at the angle of the anterior chamber.

*d.* How does the closure of the angle of the anterior

* "Glaucoma: its Causes, Symptoms, Pathology, and Treatment." (The Jacksonian Essay for 1878.) London: J. and A. Churchill.

chamber arise? The microscope shows that it is caused by the pressure of the swollen ciliary processes against the posterior surface of the iris.

*e.* Can we, then, accept Weber's explanation of glaucoma, viz., that it is the result simply of swelling of the ciliary processes? This explanation, although certainly a nearer approach to the truth than any before given, is incomplete. In the first place, what causes the swelling of the ciliary processes? Not, as Weber asserts, the presence of those maladies,—cardiac, vascular, and pulmonary—which induce general venous turgescence, for these diseases even in their severest forms are but rarely associated with glaucoma, and in many cases of glaucoma they are entirely absent. In the second place, a simple swelling of the ciliary processes would, by reason of the relation of the parts concerned, tend to displace the lens backwards and to deepen the anterior chamber, whereas in glaucoma the tendency is in the opposite direction.

*f.* Can a closure of the angle of the anterior chamber be brought about by pressure upon the ciliary processes from behind, in the absence of swelling in the processes themselves? A series of experiments upon the fresh dead eye of the pig and the human subject established the fact that a very slight excess of pressure in the vitreous chamber suffices to drive the ciliary processes forward against the periphery of the iris, and to close the angle of the anterior chamber, the closure being so complete that fluid in the anterior chamber, even when under a pressure much greater than that proper to the eye, can find no exit. This result presented a close simulation of the conditions which exist in glaucoma, viz., a slight advance of the lens in association with a complete closure of the angle of the anterior chamber. It appeared to indicate that the primary cause of glaucoma is some condition which induces a slight excess of vitreous pressure over aqueous pressure.

*g.* A variety of considerations, too lengthy to be repeated here, led to the conclusion that a current of intraocular fluid

is constantly passing from the vitreous chamber to the aqueous chamber, by osmosis through the suspensory ligament, where it bridges across the interval between the ciliary processes and the margin of the lens,—the "circumlental space." Any obstruction in the path of this current would, it appeared certain, cause an excess of pressure in the vitreous chamber, and induce a condition of things similar to that which exists in glaucoma.

*h.* Narrowing of the circumlental space suggested itself as a possible cause of excess of pressure in the vitreous chamber. The following considerations, amongst others, favoured the idea that this is actually the cause of glaucoma:—Glaucoma is pre-eminently a disease of advanced life. In advanced life the lens increases in diameter, and thereby presumably narrows the circumlental space. Injuries of the lens, which cause it to swell as a whole, frequently induce glaucoma. Swelling of the ciliary processes is undoubtedly a part of the glaucomatous process. This swelling must tend to narrow still further the circumlental space.

A definite theory as to the origin of primary glaucoma was therefore formulated in the following words :—

*The cause of primary glaucoma is a diminution of the distance which separates the margin of the lens from the ciliary processes. Two factors combine to produce this diminution, viz.:—*

1. *Increase of the diameter of the lens.*
2. *Swelling of the ciliary processes.*

*The former is usually, if not invariably, the primary change, and is itself the indirect cause of the latter.*

For a full statement of the evidence on which this theory was built up, I must refer to the chapters on pathology in my previous essay. There also will be found a discussion of the validity of the theory in relation to various points in the etiology of the disease, to the chronic and acute types, and to the curative action of iridectomy. Some facts also which are at first sight irreconcileable with the obstruction theory

of glaucoma, but which in reality tend towards its corroboration, are there considered.

The object of the present communication is to record certain recent observations which bear upon the several points in question.

Further evidence as to the escape of the intraocular fluid at the angle of the anterior chamber is, perhaps, hardly necessary, since Leber's discovery is now generally accepted. It has recently received confirmation in the experiments of Schoeler.* He experimented upon the dead human subject, and upon rabbits under chloroform and after death. Eschars were produced upon the surface of the globe by the actual cautery, and the internal pressures and the rates at which fluid escaped were measured by the manometer. No retardation of the escape of fluid occurred except when the ciliary region was the part cauterised. In the latter case a very marked retardation was invariably observed. Neither occlusion of the vortex veins nor cauterisation of the optic nerve entrance produced any such retardation. (The manometrical measurements invariably indicated an equality of pressure in the aqueous and vitreous chambers.) Again, Leopold Weiss,† by means of injections into the vitreous chamber in living animals, and the subsequent treatment of the enucleated eye with chemical reagents, has traced the transit of the injected fluid to the angle of the anterior chamber, and thence to the exterior of the globe. In a recent case of my own, high tension was set up in a blind eye by a blow. After excision, I found the angle of the anterior chamber uncompressed, but blockaded throughout the whole of the circle by aggregated blood corpuscles, the result of hæmorrhage into the chamber.

The assertion that a current of intraocular fluid is constantly passing from the vitreous to the aqueous chamber, through the circumlental space, was based, so far as my own

* Graefe's Archiv., xxv, iv. See also Lond. Medical Record, March 15, 1880, p. 114.

† Graefe's Archiv., xxv, ii,

observations went, chiefly upon evidence of a negative character. Since that time Deutschmann has pointed out* that if the aqueous chamber of the human eye be evacuated by puncture shortly after death, it is refilled within a few hours. Seeing that the same thing occurs in the freshly excised eye, the only possible source of the regenerated fluid is the vitreous humour. I have lately performed this experiment myself several times, and always with the result stated. Again, the experiments of Weiss, already referred to, indicate a current in this direction.

It will not now be denied by any experienced observer that in eyes blinded by primary glaucoma some compression of the angle of the anterior chamber is constantly discoverable with the microscope. The solid adhesion of the two surfaces which exists in the great majority of specimens is, however, occasionally absent. Of five additional specimens lately examined by myself, an adhesion is present in four. In the fifth, in which the angle of the chamber appears, under a low power, to be in a normal condition, a higher power proves beyond a doubt that the surfaces have previously been in contact, if not adherent. Cells and pigment from the surface of the iris adhere to the cornea throughout precisely that region which in most cases is the site of the adhesion. Drawing 11 exhibits a section taken from this specimen. The eye unfortunately lay for many weeks in Müller's fluid before it was opened for examination, under which treatment shrinking of the lens invariably occurs. I think that the separation of the periphery of the iris from the cornea which has occurred in this case was a *post-mortem* change produced by shrinking of the lens and traction of its suspensory ligament upon the ciliary processes. The wide separation of the iris and cornea occurred in part during the process of mounting, which in sections of this kind is unusually difficult, by reason of the slender attachments of the lens. It is worthy of notice that in this eye only of the five in question the pupil remained con-

* Graefe's Archiv., xxv, i.

sensually active for many months after blindness was complete, in fact until a short time before excision. (The case was one of very slow, painless, non-irritative glaucoma, of perhaps ten years' duration before the hyperæmia and pain occurred which called for excision.)* This suggests the inference that so long as the iris retains its consensual activity, and so long perhaps as it reacts freely to myotics, the angle of the chamber is not compressed so forcibly as to produce a firm adhesion, an inference which accords with the fact that it is chiefly during this stage of the malady that iridectomy is able to effect a permanent reduction of tension.

Again, I think it will not be disputed that the closure of the angle of the anterior chamber is directly due to the pressure of the ciliary processes against the iris, as originally asserted by Adolph Weber.† In some cases, truly, the processes are found small, retracted, and far removed from any contact with the posterior surface of the iris, but there is ample evidence to show that they have previously occupied a much more advanced position; *e.g.*, the relations of the parts in the healthy eye (Drawings 1 to 5); the invariable limitation of the adhesion to a region corresponding with these relations; my own experiments on this point;‡ the close and forcible contact, even the adhesion, of the processes with the iris discoverable in the more recent cases of glaucoma; the fact that in certain of the specimens in which the processes are greatly retracted the iris retains the impress of their previous pressure, and that detached portions of the processes remain adherent to the iris in such cases. The glaucoma sections in my own possession present the greatest differences in the size and position of the ciliary processes, from extreme turgescence on the one hand, to extreme retraction on the other: I believe, nevertheless, that the closure of the angle of the anterior chamber has been produced in every

* "Glaucoma: its Causes, &c., &c." Case 21, p. 256.

† Graefe's Archiv., xxii, i.

‡ "Glaucoma: its Causes, &c.," p. 127.

single instance by the pressure of the processes against the iris. I speak here of secondary as well as of primary glaucoma. This matter will come again under notice in a subsequent paragraph.

I come now to the examination of certain points essentially involved in my own theory. The questions on which it appeared necessary to obtain further information were these:—

1. Is it a fact that the diameter of the lens increases with age?

2. Does an increase in the diameter of the lens necessarily diminish the circumlental space?

3. Is the circumlental space actually narrower in the glaucomatous eye than in the healthy eye?

4. If so, is the narrowness due to an abnormality in the lens or in the ciliary processes, or in both?

I shall attempt to give a categorical answer to each of these questions, and to substantiate it with evidence.

1. *The diameter of the lens increases with age.*

It is easy to convince oneself of this fact by examining with the ophthalmoscope a series of cases in which iridectomy has been performed. In the eye of the child and in that of the young adult the margin of the lens is always readily visible through the gap in the iris, while in the senile eye, if discoverable at all, it is only to be seen by a much more oblique inspection, and is manifestly much more nearly under cover of the sclera than in the former case. In eyes which have been iridectomised for glaucoma the margin of the lens is almost always, in my experience, entirely removed from view. In citing this comparison I do not overlook the fact that an advance of the lens, such as generally occurs in glaucoma, tends of itself to make the margin less accessible to ophthalmoscopic inspection, or that the existence of an arcus senilis renders an eye unsuited for examination in this respect.

In order to obtain more exact information as to the size

of the lens and its relation to the ciliary processes at different periods of life, I examined ten healthy eyes (five pairs) removed shortly after death, and prepared in such a way as to reduce to a minimum any shrinking or disturbance of the parts in question.

The following was the mode of examination:—

The specimen, after lying for seven days unopened in Müller's fluid, was divided by a circular incision, corresponding roughly with the ora serrata. The anterior portion, containing the lens and ciliary processes intact, was placed in solution of hydrate of chloral 1 in 20. The remnants of the vitreous humour and hyaloid membrane were carefully removed. The diameter of the lens was measured in several meridians by means of finely-pointed spring compasses and a magnifying glass, as the specimen lay in the fluid. Some days later, during which time the lens underwent no perceptible change of size, the specimen was placed in a small covered china dish, containing just enough thick gum-water to cover it, and at once frozen solid by immersion of the vessel in a mixture of ice and salt, and then divided into halves by a meridional section, and returned to the chloral solution. The thickness of the lens was then measured with compasses. Placed with the cut surface looking upwards, the specimen was readily examined with a low power of the microscope as it lay in the chloral solution. By means of a special arrangement of the microscopic camera, and a vertical stage carrying a sheet of paper upon which the image of the object was seen projected, I made an accurate drawing, or rather tracing, of the cut surface of each specimen enlarged exactly ten diameters. The series of sepia drawings thus obtained have been reproduced, in half size, by photography, and printed in *fac simile* by the Woodbury Photographic Printing Company of 157, Great Portland Street, London. A millimetre scale was added to each of the original drawings to facilitate measurement, and to serve as a test of the accuracy of the photographic reduction. Finally, the specimens from which the drawings were made were mounted in glycerine jelly, with special precautions against shrinking, and are now preserved. Repeated examinations of the specimens at different times and under slight variations of the method of treatment lead me to believe

that a slight contraction of the lens occurs in spite of all that can be done to prevent it.

Drawings 1, 2, 3, 4, 5, exhibit the parts in question in a series of healthy eyes prepared as above described. The ages of the subjects were respectively 21, 26, 49, 65, and 90. It is manifest at first glance that in this series the size of the lens increases with age. The following table shows the actual measurements of each lens as obtained by means of compasses and a magnifier:—

| Number. | Age. | Diam. of lens. | Thickness of lens. |
|---|---|---|---|
| 1 | 21 | 8·0 mm. | 5·5 mm. |
| 2 | 26 | 8·5 ,, | 5·5 ,, |
| 3 | 49 | 8·75 ,, | 5·75 ,, |
| 4 | 65 | 9·75 ,, | 6·25 ,, |
| 5 | 90 | 9·75 ,, | 6·5 ,, |

These measurements will be found to agree very closely with those afforded by the drawings; where any discrepancy exists it must be attributed, I think, to the difficulty of ensuring an absolutely identical focus of the microscope in every instance, failing which minute variations in the degree of enlargement are inevitable. The fellow eyes of those here represented were examined also, and gave almost absolutely identical measurements in every instance. I also measured a lens which escaped intact in its capsule from an eye with necrosed cornea during the operation of excision. The patient was a man aged 29; the size of the lens corresponded closely with that in No. 2 of the above series. The difficulty of obtaining specimens has hitherto prevented me from extending this series of observations, but I think that even the small number here given, taken in conjunction with the ophthalmoscopic observations previously mentioned, afford sufficient evidence that the diameter of the lens increases with age. It would appear, further, that this increase in diameter is associated not with a general flattening and a loss of thickness, as I had previously supposed, but with an

increase of thickness: in short, that the lens undergoes a general increase of bulk with the advance of life.

2. *As the diameter of the lens increases the distance which separates it from the ciliary processes decreases: the circumlental space diminishes with age.*

It would appear possible, *à priori*, that an enlargement of the lens might, by relaxing the suspensory ligament, lead to a retraction of the ciliary processes, and that thus the circumlental space might remain unchanged in width. The drawings show that such a retraction does not occur. The close proximity of the edge of the lens, and the ciliary processes in Drawings 4 and 5, contrasts strongly with their separation in 1 and 2. From the appearances visible with the microscope, in glaucomatous eyes especially, it is evident that the apices of the processes are, to a certain extent, free to advance when turgescent, and to retract when empty, independently of the traction exercised by the suspensory ligament; and, further, by placing a specimen in glycerine and water, it is easy to prove, in the case of the dead eye at least, that contraction of the lens widens the circumlental space without any rupture of the suspensory ligament. It is probable that in every case the lens assumes a slightly more globular form after death than it has during life, and we should expect this alteration to be greater in the youthful than in the senile lens; in fact, we probably have evidence of its occurrence in those specimens in which the ciliary muscle is partly detached from the sclera by the traction of the suspensory ligament; but, having regard to the contour of the lenses in my series, it does not appear that the variation in the circumlental space can be due in any great degree to a *post-mortem* change of form in the lens.

3. *In glaucomatous eyes, after excision, the circumlental space is sometimes widely open: but there is usually evidence to show that it has previously been abnormally narrow.*

The first glaucomatous eye which I examined with regard to this point appeared, at first sight, to disprove the theory

at stake. It is represented in Drawing 7, and one side of the other half of the specimen is shown on a larger scale in Drawing 8. The circumlental space, instead of being narrowed or abolished, is here more widely open than in the healthy senile eye. It has been shown in a previous paragraph that the closure of the angle of the anterior chamber which is found in these cases is produced by the pressure of the ciliary processes against the iris. In this specimen the ciliary processes are far removed from the posterior surface of the iris, and do not reach nearly far enough forwards and inwards to account for the adhesion which here exists. The iris, nevertheless, bears unmistakeable traces of having been compressed by the processes at some previous time. In its uveal surface is a very significant concavity (see Drawing 8), limited internally by a protuberance of the uvea and by a general thickening of the whole membrane, which mark the point at which the compression ceased. The solid adhesion of the iris to the cornea is just coextensive with the compressed portion. (These details are seen with convincing clearness in the specimen itself.) Further, it must be noted that the distance between the apices of the opposite processes (Drawing 7) is greater by about 2 mm. than in any one of the healthy series; and also that the body of the ciliary muscle slopes away from the lens in a way which contrasts strongly with its prominence in every one of the healthy eyes. Now if we imagine the atrophied ciliary muscle in this drawing to be replaced by one of normal size and shape, and the retracted ciliary processes to resume a normal prominence and position, taking in each case the healthy eye as a guide, it is evident that a very much narrower circumlental space would remain; and if we go a step further and imagine the processes to be swollen, to be applied to the compressed portion of the iris, and to extend somewhat beyond the limit of the adhesion (see Drawings 6 and 12), and the lens to be pushed somewhat forward, so as to touch the iris, which it always does during life, then, certainly, the circumlental space

would be nearly, if not entirely, abolished. These supposed changes may be advantageously studied by means of a piece of tracing paper laid over the drawing.

How are we to explain the extreme retraction of the ciliary processes which is present in this and similar cases? Is it the result of the gradual suppression of their secreting function,* and of the long continued pressure upon their external surface, or is it a *post-mortem* change? Some retraction of the processes occurs probably at the moment of excision. The operation suddenly cuts off the arterial supply to the globe, and at the same time promotes the escape of blood from its interior by the division of a host of veins upon the surface, and this at a moment when the intraocular pressure is still at a high pitch; a rapid and forcible emptying of the internal vessels is the consequence. This is made manifest by the excessive injection of all the superficial vessels which occurs in glaucomatous eyes immediately after excision. This cause of retraction of the processes finds no parallel in the conditions which are present in the healthy eye at the moment of death. It occurred to me that if this explanation were the true one, the retraction might be avoided by puncturing the sclera, and thereby reducing the intraocular pressure prior to excising the eye. I carried out this idea in the next case which occurred in my practice. The specimen is shown in Drawing 6. The processes are large and prominent, and under the microscope all the internal vessels of the eye, those of the processes included, prove to be fully injected with blood. The large size of the processes appears, nevertheless, to be due rather to proliferation of the perivascular connective tissue than to distension of the vessels themselves. The difference between the processes in Drawings 6 and 7 cannot, therefore, be attributed in any great degree to the puncture in the sclera, and the consequent injection of the vessels; it has more connexion, probably, with the fact that the glaucoma was in the one case acute in character, in

* "Glaucoma: its Causes, &c.," p. 163.

the other non-irritative and chronic. A repetition of the foregoing experiment in one or two cases of the latter class would decide the question.

In these two specimens (Drawings 6 and 7) the lens now lies at some distance from the iris, but the contour of the latter shows that the lens has previously held a more advanced position in contact with it. If we imagine the lens in Drawing 6 pushed forwards against the iris, the circumlental space disappears; and supposing the processes of the right side to be advanced sufficiently to account for the adhesion at the angle of the anterior chamber, their close compression between the lens and iris is inevitable.

Drawing 12 exhibits a section from Specimen 6, in which a part of the apex of the process is thrust forward in a wedge-like form very suggestive of compression between the lens and iris. It protrudes, be it observed, not merely as far as the limit of the adhesion of the iris to the cornea, but considerably beyond it. It appeared probable that the existence of such a compression would involve some lateral swelling of the processes—some diminution of the space which separates each process from its neighbour. To test this point, I made a series of sections transversely through the processes at and near to the angle of the anterior chamber. Drawing 13 represents such a section from the healthy eye of the subject 65 years of age (No. 4). It was taken just within the angle of the chamber, so as to include a portion of the periphery of the iris, and passed very obliquely, as the drawing shows, through the ligamentum pectinatum and Schlemm's canal. In the centre are three processes cut through transversely just at the point where their uveal covering is continuous with that of the iris. Sections anterior to this point cannot be mounted, for the heads of the processes being severed from their attachments drop out; those at each side of the drawing are of course seen in oblique section. Drawing 14 shows a section from a similar or a slightly less peripheral situation in a glaucomatous eye, the specimen previously referred to (Drawing 11) in which the angle of

the chamber, though open, shows traces of previous closure; the patient's age was 70. The large size and the tortuous mutually-moulded outlines in the latter case present a significant contrast with the smaller size, the simpler contour, and the wider interspaces of the healthy processes. The tortuosity would probably have been still more extensive had the sections been made in a direction perpendicular to the surface of the globe, instead of parallel with its antero-posterior axis; they would then have passed through that part of each process which presented directly towards the lens, whereas they now pass directly backwards through that portion which lies in the groove of the suspensory ligament, and thus do not fairly represent the condition of the circumlental space. The close mutual compression which has existed at the upper part of these processes would manifestly aid in the complete obliteration of the circumlental space.

Again, in Drawing 11, taken from this same specimen, the appearances indicate with considerable show of probability that the edge of the lens has previously been in contact with the processes, its subsequent retraction having been caused by the prolonged action of the preservative solution, as explained in a former paragraph. This probability rises almost to a certainty when the section is examined under a high power. The margin of the lens between the two points of attachment of the suspensory ligament is thickly studded with cells or cell-like bodies, similar to those which coat the surface of the process.

Drawing 9 has not yet been referred to. It represents a specimen of chronic glaucoma. The course of the disease had been extremely slow. The eye had been blind for four years previous to excision; neither pain nor redness had occurred until the last three months. The onset of irritative symptoms called for excision. In the fellow eye chronic glaucoma had been permanently arrested by iridectomy. On the right side of the drawing the extent of the closure of the angle of the anterior chamber indicates a previous position of the processes which would have closed the circumlental

space. On the left side the processes are further removed from the lens, and the closure of the angle of the chamber is of much smaller extent. These relative differences are interesting as indicating the import of the proximity of the margin of the lens. In view of the slow progress and non-irritative character of the malady in this case, and in the absence of evidence that the lens has been driven forwards, it seems not unreasonable to conclude, in accordance with the appearances shown in the drawing, that the interference with the circumlental space has been less complete or less universal than in the two former cases.

Drawing 10 represents a specimen of glaucoma, the history of which was altogether exceptional. The patient, a woman, æt. 63, stated that four years ago her sight was good, when one day, while carrying a bucket of water, she felt a sudden sharp pain in the left eye, which lasted only a few minutes. Blindness was immediate and permanent. Pain occurred from time to time ever after, and set in severely three weeks ago. The other eye presented an advanced stage of chronic glaucoma, with recent irritative symptoms. The blind eye, hard, red, and painful, was excised at once; iridectomy was performed upon the other with good result. The specimen when opened showed a deeply cupped disc, proving the excess of tension to have been of long duration. The suspensory ligament, to the naked eye, was thickly coated with what appeared to be a greyish semi-opaque deposit. This proved under the microscope to be the remnant of an old hæmorrhage. Large quantities of blood corpuscles, held together apparently by a fibrinous coagulum, coated the whole of the suspensory ligament and adjacent processes; a fibrinous film containing blood corpuscles lined the cornea loosely; and corpuscles were aggregated in the neighbourhood of the angle of the anterior chamber. No trace of their natural colour remained. The ostensible starting point of the glaucoma in this case was doubtless a hæmorrhage from some part of the ciliary processes, causing sudden loss of sight; but seeing that the fellow

eye was subsequently attacked by chronic glaucoma, it is not impossible that conditions predisposing to glaucoma, or even the first stage of the malady itself, may have preceded the hæmorrhage. Whether the circumlental space was ever abnormally narrow in this eye (as indeed it now appears to be in some parts of the circle), or whether it was obstructed by the blood which everywhere thickly coats the suspensory ligament, and chronic glaucoma induced thereby, are questions for conjecture only. The specimen affords little evidence as to the point under discussion, but inasmuch as the condition of the fellow eye leads me to class it as one of primary glaucoma complicated with hæmorrhage, it cannot fairly be omitted from the series.

Adolph Weber, in his invaluable work on the cause of glaucoma, already referred to, states that it is not unusual after iridectomy to find a ciliary process apex or a pigment mass adherent to the equator of the lens, as evidence of the previous swelling of the processes. I have not yet met with a well marked example of this, but I have more than once seen peculiar changes in the transparency of the capsule at the equator, or rather just in front of the equator, which appeared likely to have been caused by the previous contact and pressure of the processes.

In spite of the difficulty of ascertaining the precise relation which the lens holds to the ciliary processes in the living eye, I think that the evidence here set forth goes far towards proving that in certain stages of glaucoma the circumlental space is unduly narrow.

4. *There is at present no sufficient evidence to show whether the abnormality of the glaucomatous eye is primarily in the lens or in the ciliary processes.*

If the substance of the foregoing paragraphs be true, it may be safely asserted that the comparatively large diameter of the senile lens is an essential factor in the production of glaucoma, or in a certain sense, that an increase in the size of the lens is the primary cause of the disease. But inasmuch as this increase is common to all senile eyes, whereas

glaucoma is an exceptional condition, we have yet to ascertain the additional factor which determines its occurrence. Excluding the case in which the circumlental space appears to have been occluded by hæmorrhage, I have as yet measured the diameter of the lens in only three specimens of primary glaucoma (Drawings 6, 7, and 9). In each of these, and especially in the two first, the lens has a diameter distinctly greater than that of the largest in the healthy series; and it is especially noteworthy that neither of these two glaucomatous lenses would lie between the ciliary processes in any one of the healthy series. This may be proved by measurement of the drawings with a pair of compasses. Should further observation show that the lens of the glaucomatous eye has, on the average, a greater diameter than that of the healthy eye at the same period of life, one starting point of glaucoma would, I think, be finally demonstrated. Meanwhile the isolated measurements afforded by my specimens will justify no general conclusion. It is not probable that the lens is of excessive size in every case. When the circumlental space in the healthy eye is reduced to such narrow dimensions as those shown in Drawing 5, a slight enlargement of the ciliary processes would suffice to establish the "vicious circle" of the glaucomatous process. Hence, I am disposed to restate the inference suggested in my previous essay, viz., that an undue size of the lens is ordinarily the predominating factor in the chronic form of glaucoma, vascular engorgement in the acute. The conditions under which such engorgement may arise, and the mode in which an excess of tension tends in any case to perpetuate and aggravate itself by inducing passive hyperæmia in the ciliary processes (Weber) were fully considered in the same work.

A critic has objected to my statement that a shallow anterior chamber is a constant feature of primary glaucoma. The objection is just, for the rule is undoubtedly not without its exceptions; and on the strength of these exceptions it must be admitted that an excess of vitreous pressure over aqueous pressure is not invariably present.

It is probable that in certain cases the lens may touch the ciliary processes and compress the angle of the anterior chamber without arresting the flow of fluid from the vitreous to the aqueous chamber; as, for example, in lateral luxations of the lens, in which manifestly the obliteration of the circumlental space at the one side involves its greater patency at the other. In like manner a slightly excentric position of the senile lens (see Drawing 9) may, perhaps, occasionally be the cause of glaucoma. In such cases no advance of the lens would occur, because a portion of the circumlental space would remain patent, and an excess of vitreous pressure over aqueous pressure would not arise. So far, then, as these exceptional cases of normal or deep anterior chamber are concerned, my description of the glaucomatous process needs modifying. A shallow anterior chamber is, however, the rule; hence a general explanation of the disease must include not merely a contact of the lens with the ciliary processes, but an obstruction to the flow of fluid through the circumlental space.

No reference has been made in the present paper to the recent researches of Brailey in the pathology of increased tension.* His discoveries are both important and interesting, and, so far as my observation has gone, find confirmation in my own specimens; but inasmuch as the changes in the blood-vessels and ciliary muscle to which he has drawn attention are, in my opinion, consequences rather than causes of increased tension, they find no place in the present inquiry. To the original and most instructive work of Adolph Weber† I desire again to express my obligation.

In conclusion, it will not be out of place again briefly to summarise the views which I have put forward in the present and in the previous essay, concerning the origin of Primary Glaucoma.

*The fundamental and essential cause of glaucoma is an*

* R.L.O. Hosp. Reports, vol. ix, parts 2 and 3.

† Graefe's Archiv., xxii, i.

Healthy
1.
Age 21
P.S.
0 2 4 6 8 10 12 14 mm.

Healthy
2.
Age 26
P.S.
0 2 4 6 8 10 12 14 mm.

3.
Healthy
Age 49
PS
0 2 4 6 8 10 12 14 mm.

4.
Healthy
Age 65
0 2 4 6 8 10 12 14 mm.

5.
Healthy
Age 90
0 2 4 6 8 10 12 14 mm.

6.
Ac. Glauc.
H 117.
Age 66.
0 2 4 6 8 10 12 14 mm.

7.
Chron. Glauc.
H105
Age 69
0 2 4 6 8 10 12 14 mm.

8.
Chron. Glauc.
H 105.
Age 69.
0 1 2 3 4 5 6 7 mm.

9.
Chron. Glauc.
III 37.
Age 69.
0 2 4 6 8 10 12 14 mm.

10.
Glaucoma.
H 283.
Age 63.
0 2 4 6 8 10 12 14 mm.

11.
Chron. Glauc.
F 174.
Age 70.
0 1 2 3 4 5 6 7 mm.

12.
Ac. Glauc.
H 117.
Age 66.
P.S.
0 1 2 3 4 5 6 7 mm.

13.

Healthy

Age 65.

0 1 2 3 4 5 6 7 mm

14.

Chron. Glauc.

F. 174.

Age 70.

P. S.

0 2 4 6 8 10 12 14 mm.

*abnormality situated in the eye itself, namely, an insufficiency of space between the ciliary processes and the lens.*

*All conditions which tend to promote venous turgescence, arterial hyperæmia, or increased secretion within the eye, may become exciting causes of glaucoma, provided that this abnormality is present.*

## ON CENTRAL AMBLYOPIA.

By George W. Berry, M.B.

That form of amblyopia in which direct vision is impaired alone, whilst that of the peripheral portions of the retinæ is entirely unaffected or but slightly less acute than normal, constitutes a group of cases which claim consideration as well from their relative frequency as from the fact that the prognosis is all but invariably good. They very rarely end in complete blindness, occasionally they remain stationary after more or less loss of direct vision, and, in the great majority of instances, they undergo partial or complete recovery. Their diagnosis is, therefore, a matter of importance, and, I cannot but think that were they always recognised as a distinct class, we should hear less of the wonderful cures produced by strychnia and electricity in amaurosis or optic nerve atrophy.

Cases of central amblyopia present themselves for treatment at Moorfields almost daily. Through the courtesy of the surgeons I have had frequent opportunity of studying them, besides having had my attention drawn to the subject at the clinic of Dr. Edmund Hansen, at Copenhagen, where they have been carefully studied for the last thirteen years, and from whence 183 cases constituted the material for an admirable treatise by Krenchel* in 1876. I would, therefore, as briefly as possible, point out what appear to me to be the chief points of importance in the diagnosis and prognosis of this form of amblyopia without giving any cases in detail. Illustrative cases will be found in Mr. Hutchinson's last paper on Tobacco Amaurosis,† and Mr. Nettleship‡ has given very accurate representations of the field of vision with reference to the central colour scotoma to which, so far as

* "Amblyopia Centralis." Copenhagen, 1876. (Abstract in Nagel's Jahresbericht for 1876.)

† Ophthalmic Hospital Reports, vol. viii, p. 486.

‡ St. Thomas's Hospital Reports, 1879.

I am aware, he was the first to draw attention in this country.

The existence of cases in which the defect of vision was owing to the presence of a central scotoma has been long known, and constitutes one of the classes of cases described by v. Graefe* in his famous lectures on amblyopia, but these were the more severe cases only in which central vision was not only relatively but absolutely worse than the peripheral. It was not until Leber† and Förster‡ discovered the means of detecting slight central defects by examining the colour sense that many cases that had come under some such category as that of amblyopia *sine causâ* were really found to belong to the central group of cases where the amblyopia was probably of toxic origin, and in which no central colour scotoma could be detected.

The first of Graefe's§ illustrative cases was evidently one of central scotoma, and probably due to tobacco poisoning, it is described as a case of the "curable form of congestive amblyopia with normal field of vision."

Central amblyopia appears in the great majority of instances to be a toxic affection most commonly caused by the abuse of tobacco. The occasional deleterious effect of tobacco on vision has long been known or rather suspected; it is referred to by Mackenzie in his well-known treatise on the diseases of the eye, and later on in 1863 by Wordsworth,‖ Hart,¶ and Hutchinson.** At that time, however, no systematic examination of the field of vision appears to have been generally practised, and consequently no thorough classification of such cases into a distinct group was possible.

The general features of these cases of central amblyopia are probably familiar to most ophthalmic surgeons. A man,

* *Vide* Zehender Klin. Monatsbl., 1865.
† Graefe's Archiv., vol. xv, part ii, p. 26, 1869.
‡ Ein Jahresbericht der Schls. Ges., 1868. Heidelberg Congress, 1871.
§ *Loc. cit.*, p. 193.
‖ Medical Times and Gazette, April 4th, 1863.
¶ Lancet, July, 1863.
** *Ibid.*, November, 1863.

generally beyond thirty years of age, complains of gradual failure in the sight of both eyes, accompanied by a disagreeable sensation of fog; usually this has been going on for some months before he applies for advice; he has seen well four or six months ago, but latterly he has not been able to read even large print, and has finally been obliged to give up his occupation. He is often alcoholic, not unfrequently has lost his appetite, or is sleepless, has suffered from diarrhœa, hæmorrhage from piles or some other drain on the system, but often he has noticed nothing unusual in his state of health; very frequently indeed he is a great smoker. There are still some ophthalmologists* who do not appear to be convinced of the toxic effect of tobacco alone in producing amblyopia, and ascribe the defect of vision more to alcohol, to which they assert patients with so-called tobacco amblyopia are always addicted. But why then should improvement take place so rapidly and so surely in many cases when the supply of tobacco consumed is diminished or altogether stopped? Again, although it must be admitted that it is very rare indeed to find one of these favourable cases of central amblyopia in an individual who smokes immoderately without at the same time being in the habit of occasionally drinking to excess, yet I have seen two typical cases, one an old man aged 70, who had been a tee-totaller for forty years, and the other a boy of 19 (the youngest patient in whom I have met this form of amblyopia), who certainly did not drink; and similar cases have been met with by others. Some cases, however, appear to be complicated with chronic alcoholic poisoning, and I think as a general rule it will be found that the more complete the alcoholism, the slower and more imperfect is the recovery on the cessation of smoking. Mr. Hutchinson is of opinion that the effect of alcohol is, on the whole, antagonistic to tobacco, and in this I am inclined to think he is right, judging from personal experience and from the experience of others whom I have questioned on the subject, and who, like myself, are susceptible to the depressing

* *Vide* Lawson's "Manual of Diseases of the Eye."

effect of tobacco in any quantity or of more than moderate strength. But we must draw the distinction between alcoholic stimulation and alcoholism, where the vital powers are actually lowered and notoriously unable to withstand any unusual strain. It is just possible then that the toxic effect of tobacco may not assert itself until the antagonistic effect of the alcohol has from over stimulation given place to a certain amount of depression and the equilibrium thus destroyed. This would appear sometimes to explain why a man who has smoked all his life with impunity is all at once affected by tobacco. Although it by no means explains the cause of central amblyopia in all cases, as on the one hand we meet with cases, already referred to, where no alcohol is used, and on the other hand a few cases occur in which the alcohol itself must be looked upon as having a more direct effect on the production of the symptoms. Anyone with any experience of smokers must have been struck by the different degrees in which different men are affected by tobacco, how some never smoke anything but mild tobacco, and seldom, except after meals, whereas others smoke the strongest tobacco at any time of the day. The fact of such idiosyncrasy being a matter of daily experience, is strongly suggestive of most cases of tobacco amblyopia being found amongst the former class. The class of patients who apply at the hospital (and it is in this class that central amblyopia is most frequent), generally smoke shag, and are often obliged to do so on an empty stomach, and although occasionally they confess to have great difficulty in learning to smoke, and being somewhat susceptible to tobacco altogether, still this cannot always be elicited.

The evidences then in favour of tobacco being the agent apart from alcohol in the production of the majority of cases of central amblyopia, are the gradual disappearance of the symptoms on the cessation of smoking without any other treatment being resorted to, and frequently without the supply of alcohol being diminished, and the occasional presence of exactly the same symptoms in individuals who do not use

alcohol at all. To these may be added the very suggestive fact of its extreme rarity in women, except in cases where there is distinct palor of the optic disc, and where the subsequent course confirms the diagnosis of progressive atrophy.

The subjects of central amblyopia often seem to suspect that their ailment is due to smoking, and express great willingness to give it up. Frequently, however, their good intentions are left unexecuted.

They invariably complain of great mistiness of vision, which varies considerably at different times, and is almost always said to be less in a dull light; they see "through a fog;" this is entirely confined to the region of the central defect, and we are frequently told that things appear much clearer when seen eccentrically. The improvement in vision in a dull light is probably only relative, that is, the difference between the indistinctness of direct vision and the clearness of indirect vision is not so marked, besides which the pupils are perhaps a little more dilated, although they are often not small even in a bright light. I have at any rate not been able to demonstrate any actual increase in visual acuity in subdued light; on the contrary, I have always found the reverse, and in one case which I examined with Masson's disc, by which means we are able to test the power of distinguishing between shades of grey differing only slightly in intensity, the light-sense was decidedly best when the absolute intensity was considerable (in bright daylight), and diminished in proportion as it decreased. This was tried with and without a stenopaic aperture (2 mm. in diameter). My observations in connexion with the light-sense in these cases are, however, too few to enable me as yet to draw any general conclusions, and, indeed, the examination of the light-sense of a definite portion of the retina is a matter of no inconsiderable difficulty. Some interesting experiments on this subject will be found in Krenchel's paper above referred to.

Usually the visual acuity has become very materially diminished before these patients apply for advice (Jæger, 14

to 20 at 8″), but we meet with all degrees, and it is just in the minor degrees, especially if there be at the same time some complication (as for instance incipient cataract or central choroido-retinitis), that there is any difficulty at all in the diagnosis. The amblyopia is generally very much the same in both eyes; when there is any considerable difference, I have, as a rule, found it to be in the higher degrees. If the acuity be as low as J. 19 or 20, it will often be found that the eccentric vision is better than the direct, and it is important to bear this in mind, otherwise an improvement may often be assumed when, in reality, the patient has only discovered how to supply the defect of his central vision by making use of the peripheral portions of the retina. Besides, according to Graefe,* when the direct vision has become very defective, an improvement in acuity amounting to double or even treble the original amount often takes place in that of the peripheral portions of the retina immediately surrounding the defective area.

The scotoma occupying the centre of the field of vision cannot easily be demonstrated in the ordinary way until the acuteness of vision is so blunted that the patient cannot make out J. 16, then it is found that within an oval area stretching from the papilla to slightly beyond the macula lutea, a white object, especially if the examination be made in subdued light, does not appear so distinct as when seen by the neighbouring parts of the retina. The long axis of the oval scotoma is horizontal, and its point of greatest saturation somewhere between the nerve and centre. Sometimes indeed it may extend further horizontally, more of the nasal part of the field being involved. In all cases, except those in which the central scotoma is the first subjective manifestation of a progressive atrophy, the periphery of the field of vision remains intact, even although the central vision should sink so low as only to be capable of distinguishing light from darkness. The peripheral colour sense is also retained as a rule, although in some severe cases it is defective; but it is never

* *Loc. cit.*, p. 215.

so completely abnormal as in the region of the scotoma. Generally, it appears to be the nasal portion of the field where the perception of green and red hues is deficient, whilst immediately above and below the scotoma it remains normally acute. Leber and Nettleship have described cases in which the peripheral colour defect has taken the form of a ring-shaped scotoma.

In the less severe cases the examination of the colour-sense affords an easier and more trustworthy test of the presence of a scotoma. Within the abnormal area there is more or less red-green blindness. Therefore, all that is necessary practically in order to determine the existence of a central scotoma to account for a certain amount of amblyopia, is to find out whether a red or a green hue is seen more distinctly outside than within the bounds of an oval area occupying the position indicated above. This is simple enough, and yet it requires some care and practice, especially if the amblyopia be slight and the patient inattentive or stupid. Each eye should be examined separately. A small piece of red paper not larger than 10 mm. square should be used. This, if held at the end of a piece of whalebone, can be brought opposite any portion of the retina whilst the patient is steadily fixing some definite object large enough and bright enough to be easily seen. If the case be pronounced, the red will appear bluish, yellowish, buff, brown, white, or of an indefinite dirty colour, according to the hue and shade taken, and the part of the scotoma on which it falls. Small green objects will give rise to much the same sensations, but it is not necessary to use more than one colour, and different hues of red are, perhaps, more easily obtainable. The colours appear brighter in shade at the very centre than between it and the blind spot, where they are often described as dirty or smudgy, and it is in this latter region that the first evidence or last traces of a colour-scotoma can be detected, and indeed the macula does not always appear to be involved. If the amblyopia be less pronounced, we should often fail to detect the scotoma if we were merely to rely upon the naming of the coloured object

by the patient, he might call it red or green whenever it chanced to fall on the retina. In such cases it is well to use two different reds, one of a somewhat bluish and the other of a somewhat yellowish hue. By bringing these alternately 8° or 9° on either side of the point of fixation, as recommended by Krenchel, it will often be found that the red appears more distinct to the nasal side, being paler, more bluish, or more yellowish-red to the temporal side. Another plan which I frequently adopt, and which is still more delicate, is to use different shades of the same red by mixing with white; the palest is then a faint pink, and with these and an average amount of intelligence on the part of the patient, the very smallest defect can readily be detected.

The scotoma is entirely negative; thus the patient on looking at a large uniformly red surface, as he recognises the colour from the impressions received by the normal portions of his retina believes he sees it red throughout, and is entirely unconscious of the altered impression caused by the scotoma in the same way as we are unaware of the want of continuity existing in our field of vision over the surface occupied by the entrance of the optic nerve. For this reason, and owing to the circumstance that the colour-sense defect in the scotoma is of the same nature as in congenital colour blindness, so far at least as there is no blindness for one colour hue without at the same time a corresponding blindness for its complement, it is possible to choose two or more colours which give the same sensation at some particular point in the scotoma. And if one of these colours be given to a surface occupying a large portion of the field, whilst only a small portion of the surface opposite this particular point in the scotoma is of one of the other colours, the impression received is the same as if the whole surface were of uniform colour. By taking advantage of this, we have another good practical way of demonstrating the existence of a colour defect which involves direct vision, namely, by the use of Pflüger's colour tests, which are based on the principle of spontaneous contrast. Thus, in five cases in which the amblyopia was not

excessive,* I have found that none of the letters on the second sheet (red) could be made out when covered with one layer of tissue paper; the sheet appeared of a faint uniform colour, like pink blotting-paper. When, however, the attention of these patients was called to the presence of letters on the paper before them, especially after they had seen those induced from the yellow and blue and from most of the greens, there appeared to be a very faint induction from the peripheral parts of the retinæ, though not sufficiently distinct to enable them to read the letters eccentrically.

As to the ophthalmoscopic appearances, I think a little too much has been made of the paleness of the temporal half of the disc. This is by no means a constant symptom, nor does it appear to stand in any relation to the duration of the amblyopia. I have recently seen a case which had lasted two and a-half years, in which the discs presented a perfectly normal appearance both in respect to transparency and colour; a certain amount of pallor is certainly frequently met with in these cases, but as much is often found without any subjective symptoms whatever, and falls quite within the bounds of physiological variation. Further, it is a well-known fact that any paleness of the discs is always most marked on the temporal side. A shallow and broad physiological cup, by no means uncommon, gives a whitish aspect to this part of the disc, and in the case where the contrast between the two halves is marked, such a condition may coexist with a slight pallor associated with the amblyopia. At any rate this appearance is in no degree inconsistent with perfect recovery, so that we cannot ascribe to it any importance in the prognosis. Of more importance, however, is an appearance occasionally met with of a sort of insidious neuritis, a local perivascularitis associated with loss of transparency and

* As I have only just procured Pflüger's tests, I have not had an opportunity of trying them oftener, but the result in the cases tried is so consistent with what we know of the nature of the affection under consideration, that I have little doubt of their applicability to all cases where the colour scotoma involves the centre.

haziness of the disc; white streaks accompanying some of the vessels just as they emerge from the nerve, and not extending much, if at all, beyond the margin of the disc, sometimes they seem to bridge over the space from one vessel to another: in short, they are the evident signs of inflammatory change. This condition is only found in the severer cases. In the great majority of cases there is absolutely no pathological change to be found with the ophthalmoscope.

In the diagnosis of central amblyopia, the presence or absence of the colour scotoma is of primary importance, should the defect of vision be slight, or any detectable complication coexist either on the part of the dioptric media, retina, or choroid. But as a general rule, the symptoms are so evident, that this test is unnecessary to establish the diagnosis; still it is well to make it a matter of routine, and with very little practice this can be done without loss of time.

The prognosis is always good as far as complete blindness goes, except in the rare cases before referred to, where progressive atrophy begins by manifesting itself as a central scotoma. A case, probably of this nature, is at present under observation.

William R., æt. 43, gave a distinct history of tobacco amblyopia twelve years ago, from which he recovered in three months, on entirely leaving off smoking. After which he "saw as well as he had ever done." On March 10th, 1880, he applied at the Hospital with much the same symptoms as had characterised his previous amblyopia. He smoked $\frac{1}{2}$ oz. of shag daily, and stated that tobacco had never agreed with him. He did not exhibit any trace of alcoholism. V = fingers at 15′ with right eye; at 8′ with left; field normal, papillæ slightly hazy, marked central scotoma for red; also defective colour vision over the whole field. On April 28th, 1880, V, left as before; right slightly improved. Both fields of vision concentrically limited, left up to 10° from point of fixation above. He had not, according to his own statement, smoked more than $\frac{1}{4}$ oz. weekly since last visit.

The cases which remain stationary, or in which the central vision continues to get worse, are generally those which have lasted some time, in which there are decided ophthalmoscopic changes, and where alcohol forms a very suspicious element, possibly also there may be in such cases some other cause which remains undetected. In the typical form where there are no distinct pathological changes to be seen with the ophthalmoscope, improvement is certain to take place on the cessation of smoking. Generally it goes the length of complete recovery, but this may take many months or even years, although I have seen vision improve from $\frac{20}{70}$ to $\frac{20}{20}$ in less than a fortnight. Mr. Hutchinson has published cases of complete blindness which he ascribes to tobacco; the fact that the subjects in question were great smokers is not enough to render this even probable, besides in two cases the discs are described as being very white.

The treatment consists in withholding tobacco and diminishing if necessary the amount of alcohol consumed. This must be insisted upon. The patients will probably also require some "mixture" to which to ascribe their cure, the nature of which may be left to the discretion of the surgeon; few drugs of the Pharmacopœia will do harm.

Little can be said with respect to the seat of the lesion in central amblyopia. Of the numerous hypotheses which have been advanced, only those of v. Graefe and of Leber appear to claim attention. According to Leber, the central scotoma is caused by a partial neuritis or atrophy of the optic nerve, or it may be merely some anomaly in the blood supply to the superficial layers of nerve fibres. Graefe on the other hand, was of opinion that the symptoms could only be explained by the assumption of some more central disturbance. In support of this latter view, we have the symmetrical distribution and equal degree of the scotoma in both eyes, the absence in most cases of any pathological changes in the retina or optic nerve, the nature of the colour blindness, and we may, perhaps, add the frequent toxic etiology of the amblyopia, as well as its curative nature. Leber's view is supported more

by the anomalous cases which further experience may prove really to belong to another category. At any rate it is by no means improbable that much the same symptoms may be produced both by a peripheral and a central lesion.

## ON A DESCRIPTION OF A NEW OPHTHALMOSCOPE.

By John Couper, M.D., F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital.*

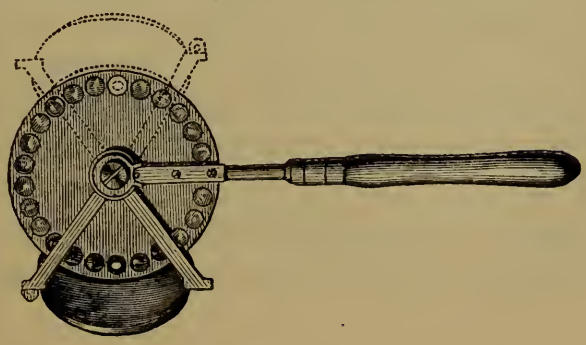
During the last few years I have modified the ophthalmoscope from time to time with a view to increase its accuracy and convenience in practice. It is unnecessary to specify the steps by which I have arrived at that form which I now present for consideration. In general terms my aims have been—1st. The utmost simplicity of parts consistent with efficiency, both as an ophthalmoscope and as an optometer; and 2nd. To avoid change of mirror or of disc, and in fact to dispense with all readjustment entailing trouble during an examination in a darkened room. Like other ophthalmoscopes aiming at completeness, it consists essentially of a concave mirror of silvered glass with a disc of lenses revolving behind it. Its chief distinctive features are—1st. The contrivance by which one mirror is made to serve all purposes, both of direct and indirect examination; and 2nd. The mechanism, by means of which each lens is so placed during direct examination that the observer looks in its principal axis.

It is superfluous to insist here on the disadvantage of creating astigmatism by an oblique position of the lens in regard to the axis of vision. The definition of the erect image is thereby diminished and exact optometry is impracticable. This defect is inevitable in the older ophthalmoscopes in which disc and mirror are fixed in a parallel position. It is obviated in some modern instruments by providing for direct examination a small special mirror fixed on the obliquely cut extremity of a short tube, and which can be placed at the angle necessary to reflect light into either eye by simple rotation of the tube. There are two objections to this plan—1st. It involves a change of mirrors. 2nd. It necessitates undue distance of the lens from the sighthole. In my instru-

ment the axis of the hinge which connects the mirror with the bracket is a tangent to the edge of the disc on the one hand, and on the other to the back of the mirror at a point close to the edge of the sighthole. The mirror is bare at this spot, in order to minimise the distance of the lens from the sighthole. Thus, when inclined at an angle with the disc, the mirror pivots on the edge of the disc, but without actual contact therewith during direct examination. On the other hand, it is made to stand parallel with the latter during examination of the inverted image by simply unbending the hinge.

By this arrangement the least practicable distance of lens from sighthole, and consequently of eye from eye, is secured. It is thus possible to use lenses of small size (of 5·0 mm. effective diameter) without optical inconvenience. A larger number of them can be set in one disc, and there is a gain in compactness. Obviously a tangent hinge alone ensures due proximity of lens to sighthole. While a hinge, the axis of which is a radius both of disc and mirror, necessitates a certain interspace between lens and sighthole which is by no means insignificant, even though the sides of the mirror be cut off to admit of sufficient tilting.

To adapt the instrument for direct examination of either eye the bracket carrying the mirror is made to describe a half circle round the disc. The mirror can thus be brought into play on either side of the disc at pleasure. This movement, which is of a simple kind, is provided with a stop-action which arrests the mirror at three several positions, viz.:—(*a*) At a point diametrically opposite the handle when the indirect method is to be employed. (*b*) On either side of the disc, as indicated by the dotted lines in the woodcut,



according as the right or the left eye is to be examined directly.

A glance at the drawing shows that during direct examination the disc must project in an outward direction considerably beyond the observer's orbit. Therefore, to avoid contact with the patient's nose, his eye must be approached from the temporal side, not from the front. All difficulties from this source are avoided by the ophthalmoscopist placing himself at the side, instead of in front, of the patient: by making the latter look outward, while he himself does the same, and by arranging that the respective heads shall be slightly inclined towards each other.

The mirror has a focal length of 8 inches and a diameter of 29·5 mm. It is no thicker than gives security against bending of the glass. The sighthole is central. Its bore is conical, the wider end being towards the observer's eye, which thus looks through a knife-edged aperture and not through a canal. The sides of this aperture have such a divergent slope that they do not reflect rays from the lamp flame into the observer's eye, and the appearance of a luminous haze, overspreading the sighthole and greatly disturbing vision, is thus avoided. With parallel sides the sighthole becomes a canal, the reflections from which may blot out the image, especially if the mirror be somewhat thick. With a conical sighthole a *thin* mirror is desirable, because the divergent sides being coated with lampblack must encroach unduly on the silvered reflecting surface if the mirror be thick. A mirror faulty in this respect is obviously valueless for examination through an unusually small pupil, only that part of its surface corresponding to the blackened sides of the bore would then reflect light into the examined eye. The thickness of the coated mirror should not much exceed 0·3 mm. The sides of the bore may then enclose an angle of 25° without any appreciable encroachment of the blackened on the silvered surface.

The diameter of the sighthole should not be less than 2·01 mm., otherwise there is stenopœic action, circles of dis-

persion are cut off, a comparatively defined image is obtained with a glass which does not measure the ametropia, and an erroneous estimate of refraction is made. Inaccuracies from this source are undoubtedly precluded by using a sighthole of 2·5 mm., and such an instrument may conveniently be used in cases where a mydriatic has been employed to dilate the pupil. It is inadmissible, however, in the case of a small pupil. With the smaller sighthole of 2·01 mm. diameter all stenopœic error is avoided if the mirror be held close to the cornea of the ophthalmoscopist.

Little need be said as to the lens series employed. The disc, which has a diameter of 41·5 mm., has 25 apertures, thus admitting of 12 concave and 12 convex lenses. For an emmetrope the following in dioptres will be found convenient:—

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| −1 | 2 | 3 | 4 | 6 | 8 | 10 | 12 | 14 | 18 | 37 | 74 |
| +1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 10 | 12 | 14 | 18 |

A myope of course requires a different series. He must enrich the concave at the expense of the convex group. He needs more concave glasses than the emmetrope, because, in the direct method, his own myopia, as well as that of the patient, must be corrected by means of the lens behind the mirror. On the other hand, he requires fewer convex lenses the greater his myopia, because the latter is employed to neutralise the hypermetropia of his patient. Generally speaking, he should contract one series and enlarge the other by as many lenses as he has dioptres of myopia. In a similar way the hypermetrope who constantly wears glasses which absolutely correct his refraction, and who, therefore, has no more difficulty than an emmetrope in holding accommodation in abeyance during the use of the ophthalmoscope, must make a converse change. His convex series must be enriched at the cost of the concave. He needs more convex lenses than the emmetrope, because his own, as well as the patient's, hypermetropia must be corrected by the ophthalmoscope, and he needs fewer concave glasses, because

in the direct examination of the myopic fundus his hypermetropia is made to serve as a measure of the myopia of the patient.

The disc has a stop action which centres each lens with the sighthole. It acts lightly enough to admit of lens after lens being brought into play by pressure of the finger on the edge of the disc without breaking the continuity of an examination. The time necessary for estimating refraction is thus materially abridged.

I have found the higher concave glasses of great practical value in high grades of myopia. Strictly speaking, the use of such glasses in the direct method is impracticable, except under two conditions, viz.:—1st. The utmost approximation of the eyes concerned; and 2nd. The observer looking in the principal axis of the lens. The size and beauty of the image, together with the perfect definition of the fine pathological changes but too often present in such cases, attest the optical correctness of the mechanism by which these conditions are fulfilled. I have thus seen the region of the macula in a myope who required, for full correction, a minus lens of $1\frac{1}{3}$ inches. Those accustomed to rely on the ophthalmoscope as an optometer and to prescribe in appropriate cases glasses from its data, will find 24 lenses too few for this purpose. To obtain a larger series in the most convenient form I have had a twin ophthalmoscope constructed which consists of two such instruments as that just described. One has only convex the other only concave lenses in the disc. The numbers are coloured red in one, white in the other disc, so that it is easy to distinguish one half of the instrument from the other in the ophthalmoscope room. By this plan a total of 48 lenses is secured, viz.:—

− 0·5, 1·0, 1·5, 2·0, 2·5, 3·0, 3·5, 4·0, 4·5, 5·0, 5·5, 6·0, 6·5, 7·0, 7·5, 8·0, 9·0, 10·0, 12·0, 14·0, 16·0, 20·0, 37·0, 50·0

+ 0·5, 1·0, 1·5, 2·0, 2·5, 3·0, 3·5, 4·0, 4·5, 5·0, 5·5, 6·0, 6·5, 7·0, 7·5, 8·0, 8·5, 9·0, 9·5, 10·0, 12·0, 14·0, 16·0, 18·0.

This series is ample enough for all purposes of exact

optometry. Each power is obtained from a single lens, an arrangement which has optical advantages over that which obtains certain powers by the superposition of lenses. My friend, Mr. Adams Frost, has added to the single instrument a small straight slide bar bearing three lenses, each of which may in turn be added to any in the disc itself. By this method no doubt an extended series of both + and − glasses can be obtained from one disc. The comparative cheapness of this plan is all that can be urged in its favour.

Under the best conditions yet devised the illumination of the fundus is comparatively feeble. If we needlessly interpose a stratum of glass with two refracting surfaces, neither of which is wholly free from optical flaws, the definition and brilliancy of the image will, *cæteris paribus,* be impaired. For precisely the same reason it is better that the mirror should have a well-constructed sighthole rather than a central spot, from which the silver has been erased, and these instruments give least definition and the dimmest image which have no perforated sighthole and with which it may chance that the rays emerging from the patient's eye encounter three strata of glass and six refracting surfaces, with the optical imperfections incidental to them, before reaching the eye of the observer.

Messrs. Pickard and Curry, 195, Great Portland Street, are the makers of the instrument. I am glad to be able to acknowledge the ingenuity and thoroughness with which they have embodied my design. The accuracy of their workmanship leaves nothing to be desired, and they produce the instrument at a very moderate cost.

## ON KERATOSCOPY.

By Litton Forbes, M.A., M.D. Pennsylvania,
*Clinical Assistant at the Hospital.*

Under the above title, Prof. Cuignet, of Lille, some years ago (1874) called attention to a method of determining the refraction of any eye by simple inspection of its cornea with transmitted light. For this purpose he made use of an ordinary ophthalmoscopic mirror, held at a definite distance from the eye under observation. The mirror was then rotated in various directions round its own axis, and the movements of the resulting lights and shadows upon the cornea were noted. From the general appearance of the bright pupillary reflex, and from the direction in which its accompanying shadow moved as compared with the movements of the mirror, M. Cuignet was enabled with more or less accuracy to diagnose at once not only the nature but also the degree of any ametropia which might be present. He was the first to systematise this method, and to show how far it might be made serviceable in suitable cases. Ten years previously, however, Mr. Bowman had already called attention "to the possibility of diagnosing regular astigmatism by using the mirror of the ophthalmoscope much in the same way as for slight degrees of conical cornea."* This suggestion would not, however, appear to have attracted the attention it deserved, and both Mr. Bowman's remark and M. Cuignet's more elaborate precepts, subsequently fell more or less into oblivion. Of late, however, the subject of keratoscopy has once more attracted attention, especially in France, while, so far as I am aware, it has not yet been touched upon by any English writer.

For some time past I have been accustomed to use the ophthalmoscopic mirror only in the diagnosis and estimation

* Donders, "Accommodation and Refraction of the Eye," English translation, p. 490.

of the degree of ametropia. The method has much to recommend it, with certain limitations and exceptions to be mentioned hereafter. Meanwhile I think it will be possible to demonstrate the following propositions with regard to keratoscopy :—

1. The existence of emmetropia, myopia, or hypermetropia, or the simultaneous presence of any two of them in different degrees in the same eye (astigmatism) may be diagnosed by simple inspection of the corneal reflex.

2. This reflex, as also the movement of the light and shade on the cornea when the mirror is moved round on its own axis, depends in low degrees of ametropia almost entirely upon the distance of the observer from the observed eye. Moreover, these appearances will vary according as a concave or a plain mirror be used.

3. The term keratoscopy is a misnomer, the form of the cornea having little or nothing to do with the resulting phenomena. These depend mainly upon the position of the retina with regard to the focal length of the dioptric system of the examined eye.

4. This method of inspection affords no means of distinguishing between axile ametropia and ametropia of curvature.

Before proceeding to consider in detail the various appearances presented by an illuminated pupil under certain conditions of refraction, a few words may be said as to the best method of making this examination. The main object should be to secure a well marked contrast between the illuminated and non-illuminated portions of the pupil, and this can, perhaps, best be done by means of a screen so arranged as to shut off all rays of light proceeding directly from the lamp to the eye under examination. The chamber in which the examination is conducted should also be as dark as circumstances will allow, inasmuch as nothing masks the phenomena of keratoscopy so much as a dull diffused light. The mirror, if a concave one, should not be held at its focal distance from the examined eye, as a small bright

circle of diffusion would thereby be produced on the retina, with the result of inducing contraction of the pupil. Supposing the mirror to be 20 centimetres focal length, it will be held most conveniently at about 50 centimetres from the patient. It should not be in the direct line of his corneal axis, but at an angle of about 15 degrees to one side of it, so as to bring the optic disc within the line of sight. In this way a more brilliant reflex will be obtained, while in cases where a mydriatic has not been used the pupil will be less liable to contract. The mirror should also be inclined at small angles, and rapidly moved through the various meridians of the cornea. This is especially important in cases of astigmatism. In low degrees of ametropia a mirror of shorter focus, say 10 or 15 centimetres, may occasionally be used with advantage. On the whole, however, it is perhaps better to use, on all occasions, the same mirror under identical conditions of distance and illumination. In this way only will it be possible to perceive those minute variations upon which the diagnosis of the degree of ametropia so much depends.

I shall now examine in detail the appearances presented by (1) the emmetropic, (2) the myopic, (3) the hypermetropic, (4) the astigmatic eye. My account of these will agree in the main with that given by MM. Cuignet and Mengin. In some important points, however, my own observations do not coincide with theirs.

If the fundus of an emmetropic eye be illuminated with light passing directly through the axis of the cornea, the following appearances will be observed. A dull red reflex will be visible over the whole field of the pupil; in the centre, however, of the field, the reflex will be somewhat dark, while around its circumference it will appear brighter and better marked. If the cone of light be next allowed to fall obliquely upon the cornea, the following changes will be noticed. A shadow which, roughly, has somewhat the form of a triangle, with its base resting almost on the pupillary edge, and its apex extending beyond the centre of the cornea, will

occupy one half of the illuminated disc of the pupil. If, for example, the light be directed upon the external portion of the right cornea, the shadow will be perceived upon the same right side, and its shape will be that of the triangle just described. This triangle will have its base directed towards the external portion of the cornea, but will not reach quite so far as the periphery. Between its base and the portion of cornea corresponding to the pupillary margin there will be a clear bright space. The apex of the triangle will be directed towards the centre of the cornea, the horizontal meridian of which might be said to bisect this angle. The remainder of the cornea will be brilliantly illuminated. If now the luminous disc formed by the reflex of the mirror be directed towards the nasal side of the cornea, the shadow will be seen to move in the opposite direction, and the same will be true according as the superior or inferior portion of the cornea be illuminated. Thus if the light be allowed to fall upon each quadrant of the cornea, by turns, the same relative position of light and shade will always obtain.

In the emmetropic eye, therefore, the shadow occupies the centre of the cornea, and extends more or less into the illuminated half. In whatever direction the mirror (concave) is moved the shadow will follow its movements. These appearances are constant in all emmetropic eyes. If distinctly seen in every meridian their presence precludes the existence of moderate or high degrees of myopia, hypermetropia, or astigmatism.

2. In the myopic eye the appearances are essentially different from those just described, and in high degrees are very characteristic. In this form of ametropia one portion of the cornea is illuminated, while the other is plunged in shade. The amount of surface, so to speak, occupied by the illuminated portion will vary directly as the degree of myopia, being always greatest in high degrees. The shadow consequently in such a case will be proportionably small in extent, and moreover will follow the movements of the mirror instead of travelling in an opposite direction. If the

mirror be turned round its horizontal axis, the luminous triangle will describe a corresponding circle around the cornea. In high degrees of myopia the apex of this luminous triangle extends considerably beyond the centre of the cornea. The shadow is, therefore, seen as a small peripheral crescent situated at the opposite edge of the pupil. In moderate degrees of myopia the bright reflex does not extend so far as the centre of the cornea. It does not, therefore, include the apex, but nevertheless occupies an appreciable portion of the illuminated pupil. In low degrees of myopia I am very sceptical, for reasons to be given further on, of the diagnostic value of keratoscopy. It is, so far as my experience goes almost impossible to distinguish such cases from low degrees of hypermetropia or from emmetropia. In describing the appearances presented by slightly myopic eyes, M. Mengin speaks of the reflex occupying but a very small surface of the pupillary edge, and assuming the form of a thin crescent placed alongside the luminous portion, and turning with it in response to the movements of the mirror; but of the accuracy of this description I have not been able hitherto to satisfy myself.

3. In the hypermetropic eye, under the condition of examination already stated, one portion of the pupillary field appears brilliantly illuminated, while the other lies in comparative shade. The shaded portion occupies the periphery of the pupil, towards the side from which the light comes. This shadow will move regularly from side to side in each meridian, or will circle round the pupil in accordance with the movements of the mirror. These movements it follows, instead of, as was the case in myopia, travelling in an opposite direction. As regards the degree of hypermetropia, it may be said generally that in low degrees the shadow occupies half or even more of the corneal surface, and has the form of a triangle, with its base at the periphery and its apex at the summit of the cornea. In moderate degrees it occupies a smaller surface, and extends scarcely so far as the summit of the cornea, assuming a distinctly crescent shape.

In very high degrees of hypermetropia it takes the form of an extremely thin crescent, deeply shaded, and surrounding the edge of the pupillary field. The hypermetropic eye is, therefore, characterised by appearances exactly the reverse of those of the myopic. In the latter the *bright portion* is situated at the periphery, on the side from which the light comes, and increases in extent in proportion to the degree of myopia. In hypermetropia the *shadow* occupies the above position, and diminishes in extent in proportion to the amount of hypermetropia present. From the intensity of the light and shade, and the contrast between them, an approximate estimate of the degree in which hypermetropia or myopia is present may be formed.

4. *Astigmatism.*—In this anomaly of refraction, as a rule, one or more meridians of the cornea are either hypermetropic or myopic, but in unequal degrees. Cases of regular astigmatism are therefore classed according as one or other form of ametropia preponderates. In mixed astigmatism both forms of ametropia are present in the same eye, though not necessarily in equal degrees. Simple astigmatism, whether myopic or hypermetropic, and especially if at all considerable, may be very readily diagnosed by means of keratoscopy. Take first a case of (Am) simple myopic astigmatism, in which the vertical meridian is myopic. Examining successively the internal and external halves of the cornea with the concave mirror, the shadow will be seen to occupy successively the same position as in a normal cornea, and its movements will not coincide with those of the mirror. Examining in the same way the vertical meridian, that portion of the cornea will be illuminated which lies towards the direction whence the light comes, and the shadow which corresponds to it will travel in the same direction as the movements of the mirror. The illuminated portion will assume the form of a crescent, or of a triangle, with its apex towards the summit of the cornea, and will be large in proportion to the degree in which myopia is present. If the crescent is small, the myopia is probably low in degree, but if it occupies half of the cornea,

or even more, in all probability at least 3 D will be required for correction. In compound myopic astigmatism (M + Am) the general appearances in each meridian will be the same so far as regards the direction in which the shadow moves, but they will differ in degree. Thus in both meridians the shade will move in the same direction as the mirror, but if the vertical meridian is more myopic than the horizontal, the illuminated portion of the pupil will be relatively larger in the vertical than the horizontal meridian. Similarly in simple hypermetropic astigmatism, the shadow will in both meridians move in a direction contrary to the movements of the mirror. In the hypermetropic meridian it will lie well at the periphery, and will be more pronounced in proportion to the amount of hypermetropia present. In compound hypermetropic astigmatism the shadow will be smaller and deeper in that meridian in which the anomaly of refraction is the most marked. In mixed astigmatism the movements and arrangement of the light and shade will vary in each meridian according to the kind and degree of ammetropia present. As regards irregular astigmatism, I have not been able to observe any constant appearances. The play of light and shade is irregular and fantastic, and does not admit of accurate analysis. The following case of regular astigmatism occurring in the practice of Mr. Streatfeild, will serve to illustrate the value of keratoscopy as an aid towards forming a rapid and approximately accurate diagnosis.

Case.—Caroline D., seamstress, aged 22, complained of difficulty in working and reading, the eyes being always more or less painful and fatigued. She volunteered the statement that she saw vertical lines better than horizontal. V in each eye without glasses $= \frac{20}{200}$.

In the right eye in the vertical meridian the shadow caused by illuminating the pupil with a concave mirror held at the distance of 2 feet, followed the movements of the mirror. The illuminated portion of the pupil was in the form of a triangle, the apex of which extended beyond the summit of the cornea.

In the horizontal meridian the shadow likewise moved in the same direction as the mirror, but was larger and not so deep as in the vertical. In the left eye the illuminated portion was fairly bright in each meridian, and occupied a large portion of the field of the pupil; the shadow here also followed the movements of the mirror. From these appearances a rough diagnosis was at once made of a considerable degree of myopia in both eyes with compound myopic astigmatism in the right, the vertical meridian being more myopic than the horizontal. Atropine was not used. With the ophthalmoscope I found for the vertical meridian in the right eye M = 4 D, for the horizontal M = 2 D. Tested for distance by means of a stenopaic slit the same results were obtained.

Vision in the right eye with spherical glasses was brought up to $\frac{20}{40}$, and with cylinders to $\frac{20}{20}$. In the left eye I found by the same methods that the vertical meridian had M = 4 D, while the horizontal had M = less than 5 D. With a spherical lens − 4 D, and a cylinder, − 0·50 D, axis vertical, $V = \frac{20}{20}$.

Did space permit other and similar cases might be recorded, in which an approximately correct diagnosis was arrived at by the same method.

When a plane mirror is used instead of a concave the resulting phenomena are somewhat different. With a plane mirror the relative positions of the bright reflex and its corresponding shadow are transposed, and therefore in emmetropia, hypermetropia, and low degrees of myopia, the movements of the corneal shadow will be in the same direction as those of the plane mirror. If the mirror be pivoted from left to right the shadow will also move from left to right. On the contrary, in moderate and high degrees of myopia it will travel in a direction opposed to the movements of the mirror. The play of light and shade obtained therefore with a plane mirror and a myopic eye will, as regards the position and movements of the shadow, be precisely the same as those witnessed in a hypermetropic eye when a concave mirror is used. The truth of this assertion can easily be demonstrated

experimentally. Theoretically, it depends upon the fact that rays of light proceeding from a plane mirror are divergent, while from a concave they can become divergent only after previous convergence and intersection. Rays of light proceeding from any mirror are necessarily refracted in their passage through the dioptric media of the eye, and go to form an inverted image of any luminous point on the retina. With a concave mirror, however, this inverted image is seen only in cases of emmetropia and hypermetropia, or very low degrees of myopia. In high degrees of myopia the inverted image becomes inverted a second time, and thus appears upright to the observer.

The play of light and shade, and the apparent movements of a shadow occupying any portion of an illuminated pupil, depend mainly in the degrees of myopia upon the distance at which the observer is placed. This may easily be demonstrated experimentally in the following manner, as suggested by M. Parent. If a glass of + 1 D be placed before an eye already known to be emmetropic, and it be thereby rendered slightly myopic, an observer at a distance of say 75 centimetres will perceive the corneal shadow moving in an opposite direction to the movements of his concave mirror. This shadow will, however, be very feeble, and will require, in order to be satisfactorily observed, a mirror of a small radius of curvature, in other words, a large circle of diffusion, together with a weak illumination, and the effect of contrast in a well-darkened room.

In an eye myopic to the extent of 1 D a real and inverted image of its fundus will be formed at the distance of 1 metre. If the observer be at a distance of 40 or 50 centimetres from such an eye he will be within the focus of its dioptric system, and will see the circle of retinal illumination in the upright image. The shadow upon the cornea will therefore travel in an opposite direction to the movements of the mirror, just as in emmetropia or hypermetropia. In proportion also as he withdraws further from the observed eye he will receive rays ever increasingly convergent. When he is at 80 centimetres

from the nodal point of this eye the convergent rays will be at not more than 20 centimetres from their point of intersection. Under such circumstances if the observer is emmetropic they will meet in front of his retina, and will form a circle of diffusion equal to that which would be formed in the case of a myopic eye of 5 D, which should fix an object placed at the distance of 1 metre. At a still greater distance, say 95 centimetres, the rays of light are only 5 centimetres from their point of convergence. The circles of diffusion are consequently still further increased, and the condition of his vision will, under the circumstances, be similar to that of an emmetropic person who attempts to see objects situated at the distance of 1 metre with a glass + 20 D before his eye. If the observer gradually withdraw to a still greater distance, the circle of diffusion upon his retina will continue to increase until, at a given moment, the real and inverted image of the illuminated circle will lie between the cornea and anterior focus of his eye, and the circles of diffusion will then have attained their maximum. Under such conditions all gradations of light and shade will be imperceptible. Nothing indeed will be visible but a uniformly illuminated circle, projected to a certain distance, and affording no keratoscopical indications whatever. Let a printed page be looked at under similar conditions, and its surface will appear of a uniform grey, the lines and interspaces forming one blurred and confused image.

If, however, the observer withdraws to a distance greater than 1 metre from an eye myopic = 1 D, he will perceive a shaded and illuminated portion when he has reached a distance of 115 centimetres, or sooner or later according to his amplitude of accommodation. But under these circumstances the shadow will no longer move, as it did before, in a direction opposite to that of the mirror. It will now move in the same direction, and a real and inverted image of the illuminated retinal circle will be visible. In low degrees, therefore, of myopia the corneal shadow will move either in the same or an opposite direction to the movements of a concave mirror,

according to the distance at which the observer is placed. It follows from this that keratoscopy can be but of little service as a means of diagnosing slight degrees of myopia.

At distances greater than one metre the characteristic appearances are difficult to seize except under peculiarly favourable conditions. For in such slight forms of myopia, the reflex being seen in the upright image, while the rays of light from it are convergent, circles of diffusion are apt to be formed upon the observer's retina. The degree of enlargement moreover being greater in M than in H, the characteristic appearances of light and shade are necessarily spread over a larger surface, and must, therefore, be less intense.

Although the term "keratoscopy," as applied to the reflex obtained from illuminating an eye under the conditions already mentioned, may be retained for convenience sake, it is clearly a misnomer. The phenomena observed depend almost entirely upon the position of the retina with regard to the focus of the dioptric system of the eye examined. In myopia the retina lies beyond, in hypermetropia within, the focal length of this system. Now rays of light proceeding from a concave ophthalmoscopic mirror held at about 50 centimetres from the examined eye, will produce upon its retina an inverted image whatever be the conditions of refraction present. This image will therefore move in exactly an opposite direction to any movement executed by the mirror. In emmetropia we have an upright image of objects at the fundus of the observed eye. The image of the flame projected upon the retina will therefore be seen in its true position, viz., inverted, and its movements will be opposed to those of the mirror. The same is also true for hypermetropia. In myopia the retinal image is equally inverted, but when seen at a certain distance, namely, beyond the punctum remotum of the observed eye, it is a second time inverted by the observer's eye. The movements of the mirror are, therefore, really inverted twice, and, consequently, the motions of the flame, the shadow, and the mirror, are all apparently in the same direction. But as already remarked, the intensity

of the shadow, or what amounts to the same thing, the brilliancy of the illuminated circle, will be proportional to the amount of ametropia present. The reason of this is sufficiently evident. In hypermetropia we obtain an upright and virtual image of the circle of retinal illumination, which image is small in proportion to the amount of H present. In myopia we obtain a real and inverted image, and the higher the degree of M the smaller this inverted image will be. In keratoscopy we see the circle of illumination in H in the upright image, and in M in the inverted, and, consequently, in proportion to the amount of ametropia present, so will the circle of illumination and its shadow be correspondingly small. Thus, in the image of the retinal reflex which is half illuminated and half in shade, the light and shadow occupying respectively a smaller space must, in accordance with a well-known law, be more intense. The converse is also true, and therefore the more nearly emmetropic an eye is the less marked will be the movement and the contrast of light and shade. This may be demonstrated experimentally, as pointed out by M. Parent. With a biconvex lens of 7 centimetres focal distance let a printed page be looked at, the lens being held so as not to form a real and inverted image of the print. At the distance of 6 centimetres (H) the characters will appear black and well defined. At 7 centimetres (emmetropia) they will appear magnified, but scarcely so well defined as at the shorter distance. At 8 centimetres (M) from the page, the lens being held close to the observer's eye, the characters become blurred and are no longer black, but grey. If the distance be still further increased, the upright image being retained, the type will become so pale and confused as scarcely to be visible.

In keratoscopy, as already sufficiently shown, the position and movements of the corneal reflex are directly correlated with the refraction of the eye observed: they can, therefore, be so interpreted as to give, within certain limits, more or less accurate information as regards any anomaly of refraction which may be present. The indications afforded apply, how-

ever, only to the sum of the refraction of all the media. No clue whatever is furnished by which an anomaly of refraction could be localised in one component of the dioptric system rather than in another. Keratoscopy is, therefore, useless as a means of differentiating between axile ametropia and ametropia of curvature. It can at most inform us as to the position occupied by the retina in relation to the dioptric system as a whole. Its indications will, moreover, be precise and trustworthy, exactly in proportion as the retina lies markedly beyond or within the focal length of that system.

In conclusion, a few words may be said as to the value of keratoscopy from a purely practical point of view. My own experience of the method does not by any means enable me to agree with the views as to its value put forth by M. Cuignet and others. As a rough and ready method of diagnosing high degrees of ametropia, whether myopia, hypermetropia, or astigmatism, it is certainly both convenient and trustworthy. In low degrees of ametropia it is neither easy of application nor can the results it gives be at once accepted as absolutely correct. Its chief value lies in the readiness with which by its means cases of mixed astigmatism may be rapidly diagnosed. It has also the advantage of rendering an observer independent of the patient's statements; but this it shares in common with the displacement of the retinal vessels and with other well-known methods of diagnosing refraction. In cases of nystagmus, in which the observation of the direct image is sometimes confessedly difficult, as also in cases of contracted pupil, keratoscopy will often be found to render good service. While, therefore neither very accurate nor of very wide application as a means of diagnosis, it nevertheless possesses a certain value when used in conjunction with other and more strictly scientific methods.

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## PULSATION IN EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

By A. Stanford Morton, M.B.,

*Senior Assistant Surgeon, Royal South London Ophthalmic Hospital.*

In embolism of the central artery of the retina, the phenomenon of a current of blood moving slowly and uniformly, or in detached discs or columns with intervening light spaces and in a jerky manner, in the affected vessels, has been referred to by various authors, and may be observed in the greater number of cases. Though, however, I have seen each of the above movements in the arteries, I have never seen them as they have been described in the veins.

The jerky onward movement is probably due to an embolus only partially blocking the calibre of the vessel affected, so that small quantities of blood may be forced past the obstruction in an irregular manner, but not in sufficient amount to fill the collapsed vessel. The to-and-fro movement is not so easily explained in this way but might be produced by a collateral vessel lending a small supply of blood, which moves in either direction in the collapsed artery, until a small quantity, escaping from the central side of the obstruction, furnishes the impetus which hurries it onwards.

The peculiar, very slowly moving, uninterrupted, onward flow of variously mixed darker and lighter shades of blood may find a possible explanation in the supposition that when the central embolism becomes impacted a small fragment is detached and carried on to the distal side of the portion in view, where, by retarding the flow of even what little blood passes the primary embolus, it gives rise to a slow stagnant current and partial separation of corpuscles and liquor sanguinis. Where the blood cylinders come to a complete stand-still, as in the case mentioned by Soelberg Wells in his

"Treatise," the second embolism has probably been complete. In some cases on record the movement of the blood is said to have been "often for a short time pulsatile," and in one which came under my notice at the South London Ophthalmic Hospital last March, when seeing Mr. Watson's patients, this symptom presented itself in a remarkable degree. The patient, a man æt. 34, came complaining of having noticed his sight "cloudy" for a week, but on covering his right eye two days previously to his visit, he found that he could not see with the left, with which he now had bare perception of large objects to his temporal side. On examination with the ophthalmoscope, I found the usual appearances of embolism, such as œdema of the retina and cherry-red spot at the macula, but the most remarkable feature was the steadily rhythmical onward pulsatory jerks of the regular blood discs or cylinders with intermediate light spaces, into which the contents of both branches of the bifurcated central artery were divided, and though sometimes these coalesced and passed on irregularly, yet several times during the observation I was enabled, by steadily fixing my attention on one blood disc, to count eleven pulsations or onward jerks during the ten seconds which it occupied in traversing the distance from the point of entrance of the artery to the periphery of the optic disc, from which position, though continuing nearly to the periphery, it was difficult to follow the pulsations closely enough to count them.

On again seeing this case, ten days later, no current or spontaneous pulsation could be detected in any of the arteries, which were all evenly filled, of fair size, and showing pulsation on firmly pressing the globe. The œdema was rapidly lessening, and the vision then or soon after was Jaeger 12. In this case the embolism, though in the central artery, must have been partial and rapidly absorbed, or the collateral circulation speedily established.

A case is reported by Liebreich, where cylinders of blood were observed to flow in a jerky manner in the vessels over a tumour between the choroid and the retina, which has a

special interest in reference to the case I have noted above, inasmuch as in it there was albuminuria present, and it might be supposed that the appearances described were due to pressure on the vessels produced by albuminuric œdema, but the fact of the symptoms being unilateral, and the obstruction posterior to the entrance of the central artery, would negative this theory.

## A SIMPLE MODE OF ASCERTAINING THE ACUTENESS OF VISION IN YOUNG CHILDREN.

By George Lawson, F.R.C.S.,
*Surgeon to the Hospital.*

All surgeons who have much to do with ophthalmic work must have experienced a difficulty in estimating the sharpness of vision in young children who do not yet know the alphabet, and whose sight cannot, therefore, be tested by any test types. I have lately used a very simple method, which seems to answer well. To quote a case :—

A young child, æt. 4 years, had a marked strabismus, and it was desirable to ascertain the sharpness of vision in each eye. I took a plate or a small *papier-mâché* tray, and sprinkled on it some mixed bird seeds, hemp, canary, millet, rape, flax, and maw seeds. I selected a hemp seed, and having covered up one eye with the hand, gave the seed to the child, and asked him to pick out a similar seed from those on the plate. When he had accomplished this task, I took a smaller seed, and then one still smaller, until at last I gave him a maw seed which will correspond as nearly as possible to a letter of Jæger 1 test types.

If the child can match the maw seed, we may fairly conclude that had he been able to read he could have read Jæger No. 1. Both eyes may, of course, be tested in the same manner. The distance at which the child holds his head from the plate whilst matching the seed given to him may also be taken into account.

Beads of different sizes would scarcely do as well, as it would be difficult for a child to compare the different sizes in beads which were all black, and coloured ones would not answer, as the child would be guided by the colour in his selection.

## HOUSE SURGEON'S NOTES—TREATMENT OF IN-PATIENTS.

By R. Marcus Gunn, M.A., M.B,
*Late Senior House-Surgeon.*

### *Gonorrhœal Ophthalmia.*

When one eye only is affected the sound eye is at once protected by the application of Buller's shield, kept in position by strips of strong adhesive plaster.*

*Treatment of the Inflamed Eye.*—Iced compress or iced alum lotion is kept constantly applied over the lids, and a solution of chloride of zinc (2 grs. to the ounce) dropped into the eye from three to six times in the twenty-four hours, the frequency varying according to the amount of the discharge. The everted lids are painted once daily with a solution of nitrate of silver (20 grs. to the ounce), and the patient is instructed to bathe the eye frequently with cold alum lotion. He takes the ordinary hospital diet with the addition of tonics when the general health demands it, and the urethral discharge is treated locally.

Under this treatment the conjunctival discharge is seldom very troublesome; but, should it collect much under the lids, syringing with cold water two or three times daily is employed.

The manner of application of the iced compress will be described later, under the treatment of congenital cataract. The addition of a little ordinary alum to the melted ice constitutes the iced alum lotion.

Should there, on admission, be decided localised corneal haze, with or without ulceration, or should such a condition appear after admission (as it sometimes will notwithstanding all precautions), cold poppy lotion is used instead of the iced compress, and drops of a solution of sulphate of eserine (2 grs. to the ounce) are dropped into the eye six times daily. The chloride of zinc drops are often discontinued

* For description and method of application, *see* The Lancet, May 16, 1874. Also Lawson, "Diseases of the Eye," 4th edition, p. 10.

or a weaker solution is employed, but even in cases of deep ulceration this solution does not cause the irritation that might be expected. The brushing of the lids is continued, great care being employed in their eversion, so as to avoid rupturing the affected corneal tissue.

When the discharge has nearly ceased the lids are only brushed once every second or third day, and this treatment is gradually discontinued. The good effects of eserine on the true, deep gonorrhœal ulcer are very decided, and even where there is a large sloughing of cornea these drops may be used with great advantage.

"Buller's shield" mentioned above affords an excellent protection to the sound eye, but great care is needed in fixing the plaster at first; and the nurse is instructed to examine its security frequently, as the constant application of fluids to the affected eye is apt to loosen the strips fixed on the nose.

Should the patient complain of slight irritation of the sound eye, with gumming of the lids, *while the plaster is still properly fixed,* the shield is not at once removed, but the condition of the eye is observed repeatedly (as can readily be done through the watch-glass); if the inflammation increases and discharge begins to show itself, the shield is at once removed, and the eye treated energetically with iced lotion, &c., as above. Very frequently, however, the symptoms of irritation and gumming accompany the use of the shield, and do not give rise to any serious inconvenience.* In such cases it is best to raise the protector from the outer side, to bathe the eye with a clean rag and alum lotion, and to apply a little simple ointment to the edges of the lid: the protector is then replaced and fixed as before.

### *Corneal Ulcers.*

The treatment of ulceration of the cornea varies necessarily according to the character of the ulcer. Where it is

* In these cases I have generally found an old history of weak eyes and gumming in the lashes. Such patients have been in the habit of rubbing their eyes in the morning and picking their lashes with their fingers, and this doubtless accounts for their liability to contract gonorrhœal ophthalmia.

deep, with an irregular, undermined periphery and surrounding infiltration, with or without hypopyon, nothing gives such good results as the use of eserine drops five or six times daily. The eye is at the same time bathed for some minutes every half hour with hot poppy fomentation, and quinine is administered internally thrice daily.

If there be much pain from the commencement, or if the eserine drops cause it to appear, bathing with hot belladonna fomentation and the instillation of atropine may be substituted; but in such cases the progress towards recovery is generally less rapid, and the result less satisfactory.

In cases of abscess of cornea, or if there be dense purulent infiltration surrounding an ulcer, a section right through the affected tissue is made with a linear knife, and the eserine treatment pursued. It is seldom necessary to re-open this wound, as in Saemisch's operation.

A seton in the temple is of benefit when there is persistent photophobia, and also in cases of vascular ulcer.

Stimulation with strong solution of nitrate of silver (applied to the part with a fine camel hair brush) is used in the indolent clear ulcer. Phlyctenular ulcers when they do not tend to perforate, and when they are not accompanied by much photophobia, are treated by insufflation of calomel, or the application of dilute yellow oxide of mercury ointment (Pagenstecher) once daily.

In all cases of ulceration a large shade is used, and the patients are kept in a darkened room.

### *Soft Cataract.*

Immediately after needling a drop or two of atropine solution is put into the eye and an ice compress applied. The latter is kept on for the next two or three days, and is then removed if there be no pain or marked ciliary injection. Atropine (4 grs. to the ounce) is dropped in six times during the day and two or three times during the night for the first two days; it is then used about four times daily. The condition of the bowels is attended to.

Immediately on the occurrence of pain, or if the globe become tense, two leeches are applied to the corresponding temple, or an ounce of blood is removed by Heurteloup's artificial leech.

If the anterior chamber is well filled with soft lens matter, or if the eye continues too tense, the swollen lens is removed on the fourth day by suction-syringe (Teale's or Bowman's) or curette. After this operation the ice compress is re-applied, atropine used frequently, and blood abstracted from the temple if there is pain.

On removing the ice compress a dry pad is kept on and the eye well shaded.

### *Senile Cataract.*

After the ordinary linear extraction a small strip of plaster is laid across the closed lids of each eye so as to prevent the possibility of the patient opening his eyes during the period of excitability after the anæsthetic. A small piece of lint and a circular pad of cotton wool are laid over each eye and secured by a cataract bandage.

At the evening visit this dressing is removed, the patient being warned not to attempt to open his eyes. Each strip of plaster is now raised from below and the lower lid gently drawn downwards so as to allow the escape of any pent-up tears which frequently collect and give rise to much discomfort. The strip of plaster is then replaced and dressing applied as before, fine linen being placed next the eyes. The condition of the bowels is inquired into, and a gentle aperient given if necessary.

Next morning the eyes are bathed gently with luke-warm water and the strip of plaster removed entirely. The lids are dried carefully with some absorbent cotton wool. The lower lid is now drawn down slightly as before and fresh dressings applied. Oil or simple ointment is rarely used for the lids, and that only when there is an eczematous condition, causing the dressing to adhere to them.

In the evening the dressings are again removed and fresh

ones applied, but there is no necessity for *bathing* the eyes at this time in ordinary cases.

This treatment is continued for the first week, when, if all has gone well, the case is put into the hands of an experienced nurse, who attends to the dressings in future. On the tenth day the cataract bandage is discontinued and the second eye left uncovered. Three or four days later both eyes are uncovered, a shade only being worn over.

The various accidents and contingencies of the after-course of the treatment are met as they arise.

*Iritis, &c.*—On the occurrence of *pain*, which cannot be accounted for by any bad position of the dressings, two leeches are at once applied to the temple. Iritis seldom appears before the fourth day, and one of its earliest symptoms is slight *itching* on the eye, oftener accompanied by a creeping sensation down the corresponding side of the nose. When such symptoms occur leeches should be applied *at once.*

Heurteloup's artificial leech is not to be recommended soon after cataract extraction, as it drags too much on the eyelids and may cause reopening of the newly-healed corneal wound.

*Indications for the use of Atropine.*—Atropine is used (1) in cases where lens-matter has been left in the eye at the time of operation; (2), when, from some difficulty in the extraction of the lens, unusual interference with the iris has been necessitated; (3), on the occurrence of symptoms of iritis. In the first two conditions the atropine instillation is commenced on the evening of the second day.

When slight iritis with lacrimation appears after the first week a blister to the temple or over the mastoid process is useful, and may be repeated every second day if necessary. Atropine sometimes causes irritation in such cases, and daturine (4 grs. to the ounce) should be substituted; or, if a stronger mydriatic is called for, duboisine (1 gr. to the ounce) may be used.

*Suppuration of the edges of the wound* generally first shows on the morning or evening of the second day, and is recognised by œdema of the lids with a slight purulent discharge on the dressing. Frequent bathing with hot poppy fomenta-

tion is now required and eserine should be dropped into the eye thrice daily. The bowels are freely opened by colocynth and mercury pill and afterwards quinine or ammonia with bark is administered. Leeches are applied if much pain be present. Under this treatment the corneal suppuration seldom runs a very severe course.*

When there has been unusual difficulty in the extraction of the lens, entailing much instrumental interference, an ice compress is applied immediately after the operation, and is kept on for the next three days, or until the risk of corneal suppuration is past.

When the lens has been extracted through a corneal wound without iridectomy (as in the Le Brun operation) eserine is dropped into the eye twice daily for the first two days or until the wound is healed. Atropine is then used occasionally, so as to prevent iritis being set up by the contracted iris coming in contact with fragments of lenticular substance.

*Secondary Operations.*—After needling opaque membrane or iridotomy for closed pupil ice compress is applied for the first two days, atropine is dropped into the eye six or eight times daily, and iritis is treated by leeching, &c., as already described. The use of atropine should not be continued too long in such cases, as occasionally increased tension comes on under its use.

*Iridectomy.*

After the operation a cold wet compress is laid over the eye and removed about five hours later, when dry dressing is applied.

In glaucoma, where one eye only is affected, eserine is dropped into the other eye three or four times daily for a few days, so as to prevent the rise of tension which not unfrequently occurs when it is left to itself. This treatment is also advisable when one eye has been *excised* for glaucoma while the other eye remains sound.

* In the last 200 cases or so under my charge I have seen no case of failure of operation due to this cause. This good result I mainly attribute to the great care taken by Mr. Burnham (then Assistant House-Surgeon) of the instruments under his charge.

## ON THE CONDITION OF THE OPTIC NERVE, CILIARY BODY, AND IRIS, IN INCREASED TENSION.

By W. A. Brailey, M.D., and Walter Edmunds, M.D.

In every case of increased tension, whether accompanied by retinal hæmorrhage or not, and whether primary, or secondary to injury, corneal ulcer, or inflammation of the uveal tract, there is found some change in the optic nerve. This in the majority of cases examined presents itself as an excessive amount of a tissue roughly resembling, though far more dense, than the ordinary fibrous stroma of the nerve.

The nerve in transverse section exhibits numerous apertures of blood-vessels, each surrounded by concentrically disposed fibrous tissue, in which is some excess of staining nuclei.

The zones thus constituted seem at first sight isolated from each other, but a closer examination shows that they are connected by thinner fibrous bands. The whole doubtless is merely the fibrous stroma of the nerve hypertrophied especially in the neighbourhood of its vessels. These are larger and far more numerous than usual, and generally show some proliferation of their endothelium. The nerve-tissue proper shows some increase of its own nuclei, and its bundles present in section very irregular shapes, owing to the encroachment on them of the connective tissue.

In the cases recited in the Curator's Report this condition is indicated by the term *Vascular Sclerosis* of the optic nerve.

In others the fibrous zones are even thicker than these, and contain in their centre only the small lumen of a blood-vessel, or even present no indication that they have ever contained one. They press upon, and leave little space for, the proper nerve-tissue. The innermost nerve-sheath is thickened, and the whole diameter reduced.

To this condition, which appears from a comparison of the different cases to be a more advanced or a more severe form than the previous, we shall apply the term *Sclerosis.*

But a certain number present nothing more than a swollen optic disc accompanied with neuritis, by which term we imply an increase of the deeply-staining nuclear bodies of the fibrous stroma of the nerve.

These are probably due chiefly to a multiplication of the connective tissue corpuscles normally existent there. It is possible, however, that some may be nuclei in the walls of newly-formed blood-vessels, for fine focussing gives occasional indications of a parallel lineal arrangement. Some may also be migrated leucocytes.

Concurrently with this neuritis there is found an inflammation in the space between the inner and outermost sheaths of the nerve. The loose fibres which are found here normally become swollen, and covered with newly-formed staining nuclei.

There is still another condition frequently seen, in which less-defined zones of translucent substance containing some nuclei, occupy the place of the dense fibrous tissue above described in vascular sclerosis.

The central artery of the nerve is also the subject of morbid changes. Its proper walls are seen to shade off into the dense fibrous tissue, which occupies the position of its sheath, and the looser tissue constituting its sheath-space, and to blend externally with the hypertrophied stroma of the nerve.

Sometimes, however, as in very early cases of increased tension, we see nothing but an increase of the vasa vasorum in size and number. This is probably the first stage, from which the others are transitions.

Sometimes, again, the sheath is densely packed with nuclear bodies, probably leucocytes escaped from the vasa vasorum in consequence of their condition. The proper wall of the artery is also thickened by inflammation.

In some cases a translucent substance, similar to that

previously described as surrounding the newly-formed blood-vessels of the septa, fills the sheath-space of the central vessels: it gradually becomes fibrillated, loses the few nuclei it possesses, and finally constitutes the dense connective tissue in which the thickened artery is imbedded, and by which it is compressed.

From a comparison of the histories of the cases, it would appear that neuritis with a distended vascular sheath constitutes an earlier stage than that with translucent exudation, that this fibrillates into the vascular sclerosis stage, till finally sclerosis of the nerve, with a corresponding condition of the walls of the central artery, is attained. These changes are passed through with greater rapidity and certainty according as the age of the patient happens to be advanced.

Sclerosis is especially seen in hæmorrhagic glaucomas, as they are always severe forms of the disease. Though Poncet described in 1873 a case of hæmorrhagic glaucoma in which he observed sclerosis of the nerve, little attention has been directed to the condition of the deeper parts of the nerve in this affection.

Buphthalmos exhibits in its nerve the same appearances as in the other forms of increased tension in young persons. These are best exemplified in the glaucoma secondary to perforating corneal ulcers.

We have no reason to believe that sclerosis can occur even as a senile change without preceding inflammation. If this be slight (as it appears to be in most cases of primary glaucoma) the disc will give no decided ophthalmoscopic indications as to the condition of the optic nerve. In the cases where we have found it well marked, as after excision for perforating corneal ulcers and injuries, the disc has rarely been visible with the ophthalmoscope.

It will hereafter be discussed as to whether these conditions of neuritis and sclerosis occur previous or subsequent to the increased tension. We may state here, however, that we are strongly of opinion that in primary glaucoma there is a pre-glaucomatous morbid change in the nerve. In the

cases of corneal ulcers and wounds we have evidence that the neuritis closely follows upon the corneal inflammation, and precedes the increased tension.

The ciliary body is also the subject of a similar morbid condition, though not with so great an uniformity of result as the optic nerve.

It may be added that the general conclusions arrived at here are in agreement with those of previous communications by one of the authors as to atrophy of the ciliary muscle (R.L.O.H. Reports, vol. ix, part 2), and the presence of enlarged blood-vessels (vol. ix, part 3). With regard to the latter condition, a stricter test for enlargement is here imposed, those cases only being thus recognized the calibre of whose arteries attains to at least twice the average normal diameter. In both papers the inflammation of the ciliary muscle was mentioned; its sclerosis, however, passed unnoticed.

In primary glaucoma the conditions most usually seen at excision are atrophy of the muscular (especially the circular) bundles, abundant dense fibrous tissue between their scanty remains, a fibrous condition of the ciliary folds, and in these either dilated and varicose capillaries, or a total absence of blood-vessels. The whole thickness of the ciliary body is reduced, it may be to even less than a third of its natural size. The iris is atrophied, more especially near its periphery. When the atrophy of the ciliary body is extreme, the pigment cells on its internal surface may be scattered and destroyed.

The longitudinal fibres of the muscle are also affected; their origin from the neighbourhood of the sclero-corneal junction instead of being of considerable thickness from without inwards is reduced to a pointed process. The diminution in size and alteration in shape of the ciliary body, due to its sclerosis and atrophy, approximate its internal surface, and more especially its apex, to the origin of the ciliary muscle, and in this movement the ciliary body necessarily carries with it the peripheral attachment of the iris.* The apex of the ciliary body is at the same time drawn backwards

* See page 93 and Curator's Report, Plate III.

towards the insertion of the muscle, which is the other fixed point.

The result of these two movements is the removal of the apex of the ciliary body to a position behind and external to that which it previously occupied, and consequently to make tense the fibres of the zonula. This agrees with what Roeder observed during life as to the extreme tension of the suspensory apparatus of the lens in glaucoma.*

Another result is that the angle of the anterior chamber of the aqueous, instead of being a little in advance of the origin of the longitudinal muscular fibres, is pulled back much posterior to it. Further, the angle instead of being rounded and open is reduced to a mere chink.

The ciliary body presents sometimes an inflamed condition, which is especially severe in the cases of glaucoma shortly following or complicated by corneal ulceration. In primary glaucoma, however, the inflammation is usually less acute. The ciliary body and iris present numerous nuclear bodies, scattered uniformly throughout their connective stroma, and especially in the immediate neighbourhood of, or even in, the muscular fibres themselves. This inflammation is, as a rule, more conspicuous in cases where the ciliary body is comparatively but little diminished in size, and in our opinion it is the cause of the subsequent atrophy.

Between inflammation and atrophy there appears to be an intermediate stage which consists in the conspicuous presence between the nucleated muscular bundles of a translucent material similar to that above described as occurring in the optic nerve.

Some cases in our possession exhibit the inflammation far more pronounced than usual. In some of them it is accompanied by much episcleritis. One case gives colour to the supposition that inflammation may travel along the track of the anterior ciliary arteries till it reaches and implicates the ciliary body. (See Curator's Report, Plate II, fig. 6.)

* Roeder, Ueber die gemeinschaftlichen Ursachen von Glaucom, &c., 1879.

This inflammation we believe to be the first stage of disease in the muscle, and, as in the case of the optic nerve, we are strongly of opinion that this condition precedes the establishment of increased tension.

As to the enlarged vessels of the ciliary region they may be found in company with any stage, but they are most conspicuous and most uniformly present in those cases in which glaucoma follows quickly on an accident (see Group III, p. 114). They certainly bear no general relation to the amount of inflammatory disturbance. The thinning of their walls is far greater than can be accounted for by their distension. They occasionally give indications of a varicose dilatation. Sometimes the arteries are not equally affected, and the same remark applies to the changes which are found in the ciliary body. They sometimes are confined to one part, the other part of the circle being unaffected.

This shows that the changes are not the result of increased tension, which would of course act equally on all parts of the circle of the ciliary body.

In cases of corneal perforation where the lens is thrust forward against the cornea, the much stretched and cystic anterior part of the retina is sometimes found lying on the internal surface of the ciliary body.

The iris has been described as nearly brought into contact with the cornea by the sclerosis and advanced atrophy of the ciliary body. The same effect can be produced by atrophy alone: it can also be produced in cases where the ciliary part of the sclerotic has yielded, the sclero-corneal junction retaining its position. In these last, however, it is rather a consequence than a cause of increased tension.

In certain cases, however, a somewhat different condition is found. In these the ciliary body is only atrophied to a partial extent, sufficient to draw back the base of the iris behind the level of the canal of Schlemm, but not sufficient entirely to destroy the rounded shape of the angle of the anterior chamber. In these cases of glaucoma the iris is still found to be applied, though not at its extreme base (which is sepa-

rated from the anterior wall of the chamber by the rounded angle), but at that part which is opposite to the canal of Schlemm.

That the iris should be applied just in this position, and neither in front nor behind it looks, decidedly, as if there were some suction of this part of the iris towards the open meshwork of the ligamentum pectinatum. The appearances in many other specimens support this supposition. But in most cases fluid is not thus entrapped in the angle of the chamber, but the iris periphery is applied throughout.

After a corneal ulcer has perforated, the iris is usually applied to the whole posterior surface of the cornea, through the application is sometimes limited to the sector corresponding to the ulcer and to the peripheral part of the iris in the remainder of its circumference.

In increased tension consequent on serous inflammation of the uveal tract the ciliary muscle and iris do not become atrophied nor is there any sclerosis. There is considerable inflammation of both ciliary body and iris, and there are also staining corpuscles in considerable numbers on the posterior corneal surface, in the ligamentum pectinatum, and round the canal of Schlemm. The choroid is inflamed and shows some evidence of increased fluid in the perichoroidal space. The nerve presents neuritis, but no sclerosis. The iris periphery is not applied, but indeed is rather widely separated from the cornea.

Sclerosis of ciliary body is most common in hæmorrhagic glaucoma, being present in 85 per cent. of the cases.

Then follow primary glaucomas without hæmorrhage with 58 per cent., and after these the cases where glaucoma has followed accidents with 71 per cent. These last cases, however, are on the average of much less duration than the others.

In glaucoma, secondary to perforating corneal ulcers under 20 years of age, the degree of sclerosis is never very great, and it is present even moderately in only 20 per cent. of the cases.

But when persons over 20 years of age are from the same

cause the subjects of secondary glaucoma, sclerosis is found greatly developed in 25 per cent., and moderately in 25 per cent. of the cases.

Sclerosis is never seen without some atrophy, far more often the amount is considerable. Atrophy, on the other hand, is often fully developed in young subjects without any sclerosis.

We believe this sclerosed condition is the cause of the cataract occurring in glaucoma, and that a similar but slight degree of this condition may be the cause of ordinary senile cataract through mal-nutrition of the lens.

The changes in the ciliary body then are strictly comparable with those in the optic nerve. Though in young subjects there is this difference, that while inflammation of the muscle usually leads to atrophy alone, in the nerve it usually ends in sclerosis. In old persons sclerosis is the goal of both conditions.

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Since the above was written more extended and exact microscopical measurements have shown that the closure of the angle of the anterior chamber of the aqueous is principally due to a contraction of the fibrous band known commonly as the ligamentum pectinatum. The contraction of the innermost layer of this draws the connective tissue framework of the iris, which has its origin here, towards the termination of the posterior elastic lamina of the cornea. That of its outermost layer draws together to a point the meridional fibres of origin of the ciliary muscle, and contracts, through its connexion with their connective tissue stroma, the ciliary processes.

The ligamentum pectinatum may also alter in position, as before explained, by the drawing back of its posterior angle due to the development of connective tissue in the ciliary body generally.

CURATOR'S PATHOLOGICAL REPORT ON

By W. A.

*Short Abstracts of the History*

THE following cases of Glaucoma have been divided into groups, the members the most recent:—

The Pathological Reports deal especially with the parts which are most *Iris, Canal of Schlemm, Optic Disc, Optic Nerve,* and *Retina.* Other changes special interest in each case are called attention to under the head of *Remarks.* used, is explained in the paper immediately preceding this. The term *Hyaline* highly refracting, containing very few or more generally no nuclei. Capil- open on transverse section. *Translucent* implies an ill-defined, almost struc- means that their walls are thicker than normal, and contain an excess of Optic Nerve. P. C. R. = Pars Ciliaris Retinæ. C. of Sch. = Canal of

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 354. Wm. R., æt. 43. He attended Mr. Hulke as out-patient 15 months ago. Then the diagnosis was anæmia of o.d. of *Left* eye. He had gout at that time. Within the last 1½ years he has had several fits, apparently epileptic. Seven weeks ago he came again with his left eye bad. It had superficial ulceration of cornea and hypopyon and T. 2. Paracentesis was done 4 weeks before excision. | Of good size and shape, much inflamed. Some sclerosis of tissue between circular fibres. | Inflamed periphery applied to cornea; lower limb involved in a corneal ulcer. | Access closed by iris. |
| No. 395. John D., æt. 46. Two months ago he attended Mr. Adams as out-patient with embolism of art. cent. ret. of *L.*, and direct aortic murmur. V = $\frac{20}{200}$ then. It had been defective 1 month. He had been drinking much of late. He comes now with T. 3, deep a.c. and no p.l. Pupillary margin irregular, with small posterior synechiæ. Has had severe pain 1 month. | A little inflamed; moderately atrophied; thickness = 0·46 mm.; slight sclerosis of tissue between circular fibres. | Periphery separated by only a narrow chink from cornea to which it has evidently been applied. At other points of ciliary circle the periphery is just adherent. | Lig. pect. and c. of Sch. somewhat inflamed. |

THE MORBID ANATOMY OF GLAUCOMA.

Brailey, M.D.

*and Pathology of Hospital Cases.*

of which are arranged in the order of duration of the disease, beginning with

commonly the subjects of the greatest morbid change, *e.g.*, the *Ciliary Muscle*, are indicated under the head of *Other Pathological Conditions.* Any points of The sense in which the terms *Neuritis*, *Vascular Sclerosis*, and *Sclerosis* are is applied to blood-vessels when their walls are thick, glassy-looking, and laries of this description are usually larger than normal, and stand widely tureless semi-transparent substance. *Hypertrophied*, as applied to arteries, nuclear bodies. A. C. = Anterior Chamber. O. D. = Optic Disc. O. N. = Schlemm. P. L. = Perception of Light.

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Shallow central pit. Margins swollen. | Moderate vascular sclerosis. | Distended with red corpuscles. Sclerosis of adventitia of arteries of papilla and obliteration of what has been described as the lymph-space. | Considerable episcleritis. | Previous white atrophy of disc. *See History.* |
| Moderately enlarged. | Shallow central pit. | Much neuritis. | Walls hypertrophied and containing excess of nuclei. | A little episcleritis. The vitreous contains some small blood spots apparently from cil. processes below. | Previous cardiac disease and embolism. *See History.* (?) Glaucoma hæmorrhagicum. |

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 401. Amelia C., æt. 69. 3 months ago while in bed with pneumonia an acute attack of glaucoma of the *Left* came on with severe neuralgia of teeth of left upper jaw. T. 2. | Very thin, much structureless substance between bundles of muscle fibres now just fibrillating. | Periphery adherent. | Access closed by iris. |
| No. 204. Wm. B., æt. 60. Cataract extraction 3 yrs. ago, followed by some iritis. Eye needled 3 months later. Vision fair till 3 months ago, when glaucoma set in. Iridectomy 2 mons. ago, but T. not relieved. Iridectomy again 2 weeks ago in opposite direction, followed by panophthalmitis. | Slight atrophy; 6 mm. Some sclerosis of antero-internal part of muscle. Great numbers of pus-corpuscles on ciliary body and in vitreous. | Completely removed by iridectomy. Abundant pus in ant. chamber. | Lig. pect. somewhat inflamed, possibly preventing access. |
| No. 241. Jane W., æt. 55. Glaucoma of *Left* 5 mons., beginning with neuralgia of frontal region and bridge of nose; photophobia and lacrimation. At excision T. 2. Lens dislocated; upper edge in a.c. just within pupillary margin. History of a blow some time previous to the failure of sight. | Great atrophy and sclerosis as above. | Atrophied; periphery adherent to cornea. | Access closed by iris. |
| No. 88. Woman, æt. 60. Glaucoma of about 5 months' duration. History very defective. | Extreme atrophy; translucent stage of sclerosis. | Periphery adherent. | Access closed .. |
| No. 763. John James, æt. 54. Eye dim 5 months. Both sugar and albumen were found in his urine 4 months ago. Had noticed when his eye was first dim that he passed much water. Shortly before excision albumen only found, and in small amount. At excision neither albumen nor sugar. | Some atrophy and inflammation; translucent stage between muscular fibres and in ciliary processes. | Periphery adherent. | Access closed .. |

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Much enlarged; walls of arteries extremely thin. | Moderately deep cup, extending only over half its area. | Neuritis and increased blood-vessels; structureless substance filling their sheaths. | Specimens not good. | Sclerosis of blood-vessels of choroid, especially of adventitia. | .. .. |
| Slightly enlarged. Walls thick. | Swollen. Blood-vessels of papilla have their walls a little sclerosed. | Moderate vascular sclerosis, more advanced than in papilla. | Some have hypertrophied walls. Others have their positions occupied by a round mass of nuclei which fill the lumen. | Increase of nuclei in nerve-fibre layer of retina, and on inner surface of ciliary body. Choroid containing some diffused increase of nuclei, and is somewhat œdematous. | Diffuse small hæmorrhages in retina. Much episcleritis. |
| Not enlarged. | Centre of lam. crib. pushed back, its concavity filled by nuclear tissue. | Sclerosis. Strong bands are seen running longitudinally. | Walls hypertrophied. | Choroid has slight occasional patches of inflammation, somewhat like plastic. | .. .. |
| Not enlarged. | Central pit, lam. crib. pushed back into a curve. | Neuritis, translucent substance in perivascular spaces. | (?) | .. .. | Specimen not from Moorfields Hospital. |
| Enlarged .. | Very shallow cup, occupying $\frac{2}{3}$rds. of o.d. Lam. crib. sclerosed. | Neuritis, translucent substance round vessels. Lam. crib. thick and translucent looking. | Walls of smaller retinal arteries hyaline and thick. | Slight episcleritis. | Albumen, but no retinal hæmorrhages. Apparently no cardiac disease. Urine not examined microscopically. |

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 151. Ann J., æt. 55. She attended Mr. Streatfeild as out-patient 13 years ago, then diagnosis was old choroido-retinitis; she read J. 2 with + $\frac{1}{12}$. Ten months ago acute neuralgia of *R.* eye and whole *R.* side of head came on. The eye was much inflamed. P.l. soon went. In 7 weeks inflammation of eye subsided. Pain continued, though less severe, till excision. | Moderate atrophy, good shape, abundant sclerosis. | Periphery adherent. | Access closed .. |
| No. 406. Henry T., æt. 49. Glaucoma of *L.* 11 months. T. +. Staphyloma at inner side of o.d. Choroiditis disseminata at macula. *R.* myopia = 13 D. | Fair size, moderate sclerosis of circular fibres. | Periphery widely free from cornea. | Open .. .. |
| No. 144. John D., æt. 58. Glaucoma for 1 year. Painful 9 months ago. Cataract and no p.l. since 6 months. | Atrophied; inflamed. | Periphery adherent. | Access closed .. |
| No. 441. George S., æt. 73. *R.* failing slightly 2 years. Severe neuralgia of *R.* brow 8 weeks ago. P.l. ceased then. | Atrophied; sclerosis of muscle and cil. processes. | Periphery adherent. | Access closed .. |
| No. 466. Joseph B., æt. 68. Eye has failed since 2 years. The cataractous lens was removed from it 13 months ago while the T. was still in excess. Iridectomy was done upwards then. Tension soon recurred, and continued till excision, bulging the iris forwards. | Inflammation and moderate atrophy. | Periphery adherent. | Access closed below by iris, above by inflammatory exudation. |
| No. 74. Margaret H., æt. 71. Eye faded gradually without pain 3 years ago till no p.l. Severe pain 2 years ago, relieved by iridectomy. Pain recurred 1 year later, and continued more or less till excision. | Much atrophy; some inflammation; much translucent substance between fibres. | Periphery adherent, but limb gone at upper part of cil. circle. | Access closed .. |

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged .. | Shallow universal cup. Lam. crib. sclerosed. | Sclerosis in bands. Slight neuritis between them. | Some have their walls hypertrophied. Others are mere fibrous cords. Many have much irregular black pigment in their sheaths, and perhaps in their lumen. | In some places the ch. and ret. adhere, and the pigment epithelium is scattered and destroyed. There is an accumulation in retina (q.v.). | .. .. |
| Not enlarged. | Characteristic of myopia. Not cupped. | Vascular sclerosis, ill-defined. | Walls hypertrophied. | Adhesion in various places between ch. and ret. obviously from old ch. diss. | Extra deep a.c. |
| Enlarged .. | Deep cup. .. | Neuritis .. | Walls a little hypertrophied. | A little general increase of nuclei in choroid. | A.c. fairly deep at centre. |
| Enlarged .. | Pit-like cup, not up to margin. | Neuritis and vascular sclerosis. | Apparently normal. | .. .. | .. .. |
| Not enlarged. | Pit not up to margin. | Neuritis .. | No specimen .. | Some nuclei in sheaths of arteries of papilla. | .. .. |
| Not enlarged. | Shallow cup. Lam. crib. sclerosed. | Neuritis, faintly fibrillated translucent substance surrounding blood-vessels. | No specimen .. | .. .. | .. .. |

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 35. John C., æt. 44. *R.* faded gradually and without pain 3½ years ago. No p.l. one year later. *L.* failed in same way 1½ years ago, but became painful later. Iridectomy in *R.* 4 months before excision. | Atrophied, inflamed in translucent stage of sclerosis. | Inflamed somewhat; periphery adherent. | Access closed .. |
| No. 746. David S., æt. 39. *L.* faded painlessly after slight accident 15 years ago; last saw light 2½ years ago. Pained him first 3½ years ago. | Atrophied, inflamed, early stage of sclerosis. | Periphery adherent. | Access closed .. |
| No. 474. John S., æt. 68. Glaucoma subacute 4 years, recent pain. Soft leucoma of cornea from ulcer, apparently more recent. | Extreme atrophy; much translucent substance between fibres and in ciliary folds. | Much atrophied; periphery adherent; apex entangled in leucoma of cornea. | Access closed .. |
| No. 368. Naomi E., æt. 65. Sight of *L.* began to fail 4 years ago with pain and lacrimation. No p.l. since 1½ years. Severe pain lately. Soft leucoma of cornea, apparently recent. *R.* glaucomatous 2½ years. Now T. 2 and posterior synechiæ. | Atrophy; much sclerosis. | Periphery adherent; lower limb in leucoma. | Access closed .. |
| No. 64. Eleanor B., æt. 49. Bad off and on 8 years. Severe symptoms 2 years. No p.l. for 1 year. | Structure much atrophied and inflamed. Some translucent substance between its fibres. | Inflamed; periphery adherent. | Access closed .. |
| No. 178. Stephen G., æt. 41. *R.* failed quickly 10 years ago. No pain till 5 weeks ago. Attempted iridectomy 3 weeks later. | Fair size and shape; much inflammation. | Inflamed, periphery adherent. Uveal pigment rolled into view by the contraction of some inflammatory material on its front surface. | Access closed .. |

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Deep cup, with excavated sides. | Neuritis, with new blood-vessels. It is in the translucent stage of sclerosis. | Neither hypertrophied nor hyaline. | .. .. | .. .. |
| Enlarged .. | Deep cup .. | Neuritis, translucent substance round blood-vessels. | Neither hypertrophied nor hyaline. | .. .. | .. .. |
| A little enlarged. | Very shallow cup. | Neuritis, much translucent substance around blood-vessels. | No specimen .. | Sclerosis of sheaths of blood-vessels of choroid. | .. .. |
| Not enlarged. | Shallow cup. | Vascular sclerosis, a much earlier stage than in muscle. | No specimen .. | .. .. | A much more advanced stage of sclerosis in muscle than in nerve. |
| Enlarged .. | Moderately deep cup. | Much neuritis, nuclei in perivascular space, and some translucent substance. | Arteries have rather hypertrophied walls. Capillaries have walls hyaline and gaping. | .. .. | .. .. |
| Enlarged, full of blood corpuscles. | Deep cup. Abundant translucent substance in sheath of central artery. | Much neuritis, very early stage of sclerosis. | Neither hypertrophied nor hyaline. | .. .. | .. .. |

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 376A. Sarah H., æt. 70. Old glaucoma. T. 2. Iridectomy done 8 years ago with some benefit, but T. recurred after 1 year. | Much atrophy and much sclerosis. | Periphery adherent to scar of iridectomy and to cornea. | Access closed .. |
| No. 353. Sarah H., æt. 68. Subacute glaucoma many years. Attempted iridectomy upwards 10 days before excision. Traumatic cataract. Bulging cicatrix with lymph, &c. | Moderate, recent inflammation. Sclerosis. Inflammatory exudation on internal surface of ciliary body. | Periphery adherent, inflamed. | Access closed .. |
| No. 340. Woman, æt. 56. Glaucoma of doubtful duration. Iridectomy a few weeks before excision. Sympathetic inflammation of uveal tract of other eye. | Fair, sclerosed. Nuclei on internal surface of ciliary body. | Thick, with inflammatory new formation in scar of iridectomy; periphery applied. | Access closed .. |
| No. 197. Solomon S., æt. 54. Old glaucoma of *L.* It has been painful for 2 years. Has had iridectomy in three different directions without relief of T. At excision T. 2. | Very much atrophied, moderate sclerosis. | Atrophied limb; removed from some sections by the operation. | Access closed by adherent base of iris. |
| No. 86. Mary B., æt. 63. *L.* faded 30 years ago. No p.l. in 1 year. Recent pain and inflammation, with onyx and hypopyon. At excision T. +. | Complete atrophy; much inflamed. | Periphery adherent, atrophied, much inflamed. | Access closed by adherent base of iris. |
| No. 67. Charles R., æt. 55. Chronic glaucoma of many years' duration. | Atrophied, inflamed; some translucent substance. | Periphery adherent, atrophied. | Access closed by adherent base of iris. |

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged .. | Shallow cup. | Marked sclerosis. | (?) | Optic nerve shrunk to half its proper size. Sclerosis of outer walls of blood-vessels of choroid. | .. .. |
| Not enlarged. | Bad specimen | Marked sclerosis. | Neither hypertrophied nor hyaline. | | .. .. |
| Not enlarged. | No specimen. | Marked vascular sclerosis. Dense connective tissue surrounding central artery and vein. | No specimen .. | Inflammation of choroid, plastic in character. | The nerve is shown in transverse section, and is more densely sclerosed than the muscle. Not from Moorfields Hospital. |
| Enlarged .. | Deep cup .. | Vascular sclerosis. | Some have their walls thickened and hyaline. | .. .. | .. .. |
| Not enlarged. | Shallow cup. | Much neuritis. | Large tracts where choroid and retina atrophied, and adherent. Walls of blood-vessels neither hypertrophied nor hyaline. | .. .. | .. .. |
| Enlarged .. | Much nuclear tissue in front of concave lam. crib. | Very much neuritis, some ill-defined translucent substance. | Walls of blood-vessels as above. | Long ciliary arteries are large. | .. .. |

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 5. Elizabeth B., æt. 56. Glaucoma of many years' duration. Pupillary margin irregular, with posterior synechiæ. Cornea is leucomatous at one place. | Atrophied, much inflamed; much early sclerosis. | Periphery adherent. | Access closed by adherent base of iris. |
| No. 265. Man, æt. 72. *R.* cataractous 20 years, but glaucomatous only 6 months apparently. Now T. 3. Pain. | Moderately atrophied; much sclerosis. | Periphery free. It has apparently never been applied. | Open .. .. |

The following two cases must be added, agreeing as they do in all important of their symptoms and morbid appearances, which would cause them probably

| | | | |
|---|---|---|---|
| No. 452. Rebecca B., widow, æt. 56. History imperfect on account of the insanity of the patient. There is much iritis and episcleritis. The aqueous is turbid, and the a.c. in its centre is of fair depth. T. 2. | Atrophied and much inflamed. | Inflamed, atrophied, periphery adherent. | Access closed; lig. pect. and c. of S. much inflamed. |
| No. 492. Henry C., æt. 19. *R.* faded gradually 9 years ago; inflamed 2½ years. At excision T. 2. A.c. of good depth. | Atrophied and much inflamed. | Inflamed, atrophied, periphery adherent. | Access closed; lig. pect. inflamed. |

*uncomplicated by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged somewhat. | Deep cup .. | Great neuritis; some sclerosis of one side of nerve in especial. | Walls of blood-vessels as above. | .. .. | .. .. |
| Not enlarged. | No specimen. | Vascular sclerosis, pushing aside nerve bundles somewhat irregularly. | Walls of blood-vessels as above. | .. .. | Not excised in Moorfields Hospital. Transverse sections of O.N. |

pathological points. They differ only in the more inflammatory character to be ranked clinically under the head of Inflammatory Glaucoma.

| | | | | | |
|---|---|---|---|---|---|
| Not enlarged. | Shallow universal cup. | Neuritis and commencing vascular sclerosis. | Walls of arteries hypertrophied and dissociated. | Cells of pigment epithelium of retina destroyed. Pigment increased, black and scattered; outer granules and rods and cones gone. Much episcleritis following wall of anterior ciliary artery to muscle. | Patient, a stout healthy-looking woman, has been in asylum 11 years, on account of dementia. No history of syphilis known. |
| Not enlarged. | Deep cup .. | Neuritis and commencing vascular sclerosis. | Retinal arteries are represented by strong fibrous cords, mostly impervious. | Pigment epithelium as above. Episcleritis. | No history of syphilis or any serious illness. |

GROUP 2.—*Twenty-one cases of Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 483. Edward E., æt. 46. *R.* failing since 6 months. Three months before excision attended Mr. Tay, whose diagnosis was *R.* Retinitis with hæmorrhages. V. = shadows. Numerous hæmorrhages near macula and small white spots also. *L.* V = $\frac{2}{20}$. Has had gout lately. *At excision* T. 3. A.c. good. | Atrophied, thickness = ·36 mm., much inflamed. | Inflamed, periphery adherent. | Access closed .. |
| No. 777. Amelia D., æt. 50. *R.* old blind eye. Blow 6 weeks ago. Since then painful. Glaucoma probably of longer duration than 6 weeks, judging from the optic disc. History more fully given in vol. ix, p. 398. | *Upper part*, much atrophy, some inflammation, abundant translucent material just fibrillating. *Lower part*, less atrophy and inflammation, otherwise as above. | *Upper part*, periphery adherent. *Lower part*, periphery just adherent. | Access closed .. |
| No. 177. Elizabeth R., æt. 79. Glaucoma of *R.* of 8 or 10 wks.' duration. *L.* normal. History more fully reported in vol. ix, p. 394. | Of fair size, inflamed, much translucent material near centre of muscle. | Of good size, separated by narrow chink from cornea. | Access probably closed. |
| No. 364. Joseph R., æt. 40. *L.* eye quite well till 9 weeks ago, then shooting pain. Came here 3 weeks ago. Then bulging of lower part of cornea, upper part tilted upwards and pretty transparent. History more fully in vol. ix, p. 419. It is now thought that the corneal change is secondary to the glaucoma. | *Upper part*, mod. atrophy and sclerosis of tissue between muscular bundles. *Lower part*, much atrophy and sclerosis. | *Upper part*, periphery just adherent. *Lower part*, atrophied, and much adherent to the bulged cornea. | Access closed .. |

*accompanied by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Shallow central pit; translucent substance in sheath of art. centralis in nerve. | Neuritis, translucent substance round numerous small blood-vessels far back. | Walls hypertrophied. | Hæmorrhages numerous, and mostly capillary in intergranular layer. | No albumen in urine. No cardiac bruit. Had an appearance as if of excess in alcohol. |
| *Upper part* somewhat enlarged. *Lower part* of normal size. | Cupped .. | Neuritis, abundant translucent material within lymph-sheaths of blood-ves-vessels near papilla. | Arteries have hyaline walls. | .. .. | Changes in the upper ciliary region much more advanced than in lower. |
| Somewhat enlarged. | Shallow depression of disc. | Neuritis especially in front, marked vascular sclerosis. Sclerosis of adventitia of cent. artery. | No specimen .. | Choroid healthy, but its epithelium, as is not unusual, in a colloidal condition. | Changes more advanced in optic nerve than in cil. muscle. |
| Not enlarged. | Shallow pit.. | Vascular sclerosis. | Walls hypertrophied. | Hæmorrhage most in intergranular layer. | Some yielding of the ciliary portion of the sclerotic above; the region of sclero-corneal junction has not yielded. |

GROUP 2.—*Twenty-one cases of Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. | |
|---|---|---|---|---|
| No. 122. Sarah P., æt. 53. *L.* watered 9 months ago. Came dim 3 months later. Came here 4 months ago; then albuminuria; + T. and severe pain. Hæmorrhage in retina. Iridectomy upwards, followed in 3 days by iridectomy downwards. Pain relieved for 3 weeks. Then recurred, and sclerotomy done with temporary relief. | Moderate atrophy, inflamed. | Inflamed, periphery adherent. | Access closed | .. |
| No. 61. Eliza B., æt. 25. *L.* glaucomatous 9 months at *excision*. Eye enlarged. Numerous ciliary staphylomata. Abundant dark blood between ch. and det. ret. A.c. of good depth. This case is mentioned in vol. ix, p. 410. | Structure thin and inflamed. | Inflamed, periphery adherent. | Access closed | .. |
| No. 49. Stephen H., æt. 73. *L.* faded imperceptibly 9 months ago; pain came 2 months ago. Radial artery atheromatous. Pulse slow. | Atrophied and sclerosed (translucent stage). | Inflamed, periphery adherent. | Access closed | .. |
| No. 160. Jane R., æt. 38. *L.* came bad 12 months ago. Severe pain for last 4 months. At excision T. 3. Large staphyloma involving a large extent at upper and outer part of equator. This case is mentioned in vol. ix, p. 410. | Atrophy, inflammation, and very early sclerosis. | Of fair size, base adherent. | Access closed | .. |
| No. 270. Mary S., æt. 59. Pain in *R.* 14 months ago; lost p.l. in 4 months. Noticed white speck on cornea 4 months only. Severe pain for last 4 months. *L.* a little watery, but otherwise not affected. | Thin sclerosis of circular fibres: somewhat inflamed. | Inflamed periphery adherent. | Access closed | .. |
| No. 143. Sarah D., æt. 78. Attended here 5½ years ago for an accident followed by leucoma of cornea of *R.* There appears to have been no tension till 18 months ago, when it came bad with pain and lacrimation. *L.* bad 6 months; pain 3 months; now T. 1. | Inflammation of radial fibres, especially circular atrophied, with abundant early sclerosis. | Periphery adherent. | Access closed | .. |

*accompanied by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Shallow cup. | Neuritis .. | No specimen .. | .. .. | Albumen in urine. A.c. filled with blood after the sclerotomy, which was very slowly absorbed. |
| Not enlarged. | Lam. crib. pushed back, cavity filled by nuclear tissue. | Neuritis, with new blood-vessels and early sclerosis. | No specimen. Ret. detached | General diffused nuclear increase in choroid, especially near staphyloma. Some episcleritis. | Staphyloma of sclerotic in ciliary region. |
| Enlarged .. | Shallow cup. | Neuritis and some vascular sclerosis. | Walls of arteries are hyaline. | .. .. | Changes in muscle more advanced at upper than at lower part of cil. circle. |
| A little enlarged. | A deep pit .. | Neuritis and early vascular sclerosis. | ? | Diffused increase of nuclei in thickened ch. near optic disc. | Staphyloma of sclerotic at upper ciliary region. |
| Enlarged, full of blood corpuscles. | No specimen. | No specimen. | Arteries hypertrophied. | Dense leucoma of lower and outer part of cornea, with anterior synechia. A.c. exists elsewhere. | The leucoma appears to be secondary in time to the glaucoma. |
| Enlarged .. | Deep cup .. | Neuritis, early stage of sclerosis, with scattered nuclei. | Walls of arteries hyaline. | Dense leucoma of cornea. A.c. fairly deep. | .. .. |

GROUP 2.—*Twenty-one cases of Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 289. Rebecca D., æt. 66. Glaucomatous attack in *L.* 2 years ago, with acute pain. *R.* T. 2 and pain. History more fully in vol. ix, p. 393. | Extreme atrophy and sclerosis. | Periphery adherent; iris sclerosed. | Access closed, and canal closed also. |
| No. 259. Mary S., æt. 74. *R.* faded quickly with pain 2 years ago. *L.* dim occasionally. Pupil sluggish. T. 1. | Extreme atrophy and sclerosis. | Sclerosed, periphery adherent. | Access closed .. |
| No. 786. Harriet G., æt. 62. *L.* glaucoma 2½ years. The sight went very quickly, with inflammation and pain. T. 2 now. *R.* somewhat defective, but T. n. | Atrophy and sclerosis. | Periphery adherent. | Access closed .. |
| No. 382. Eliza B., æt. 65. Attended here as out-patient 5 years ago, when the diagnosis was *Retinitis with hæmorrhages* in *L.*, and a trace of albumen was found in her urine. Six months later she had several slight attacks of hemiplegia, after each of which the V. was worse. Severe pain in *L.* 2 months. At excision of *L.*, systolic cardiac murmur. *R.* about normal. | Much atrophy and sclerosis of circular fibres and ciliary folds. | Periphery adherent. | Access closed .. |
| No. 425. Isabella D., æt. 64. *R.* Graefe extraction 8 years ago; fair result, but blow soon after discharge. Soon after this, T. 1. *At excision*, T. 3; cornea hazy. Iris bulged forwards; some blood in a.c.; pain. *L.* Graefe extraction 1 year before *R.*; did well; V. now good, but T. 1. | Atrophy, much sclerosis of muscle. | Sclerosis, periphery adherent, limb gone above. | Access closed .. |
| No. 167. George J., æt. 67. *L.* chronic glaucoma. No p.l. for 1 year. Iridectomy 5 weeks ago. Blood in a.c. first observed 3 days before excision. T. 3, severe pain lately. *R.* chronic glaucoma. Iridectomy 5 weeks ago. Now a.c. shallow. T. 1 (?) | Sclerosis, moderate atrophy. | Periphery adherent. | Access closed .. |

*accompanied by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged, especially at lower part. | Moderate deep cup. Sclerosis of walls of blood-vessels of papilla. | Some neuritis and vascular sclerosis. | No specimen .. | Retina detached. Elongated thinned tract of sclerotic crossing inner equator. Anterior part of retina a little cystic. | .. .. |
| Not enlarged. | Shallow cup . | Much vascular sclerosis. | Walls moderately hypertrophied. | Retina detached. | .. .. |
| Enlarged .. | No specimen. | No specimen. | Varicose dilatations of capillaries of retina. | .. .. | .. .. |
| Enlarged .. | Shallow cup, sclerosis of arteries in papilla. | Vascular sclerosis. | Walls hypertrophied. | .. .. | Diagnosis 5 years before excision was *L.* Hæmorrhagic Retinitis, with a trace of albumen. |
| Not enlarged. | Cupped .. | Enormous degree of vascular sclerosis. | Walls of arteries and capillaries are hyaline. | Long internal ciliary artery is enlarged. | Old cataract extraction. |
| Enlarged .. | Slightly cupped. | Vascular sclerosis. | Hyaline thickening of walls of smaller arteries. | Abundant hæmorrhages in intergranular layers. | .. .. |

GROUP 2.—*Twenty-one cases of Glaucoma,*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 290. D. W., æt. 71. *R.* failed gradually 20 months ago. At excision some pain. T. 3; barest p.l. *L.* irritable. | Atrophy and sclerosis. | Periphery adherent. | Access closed, but canal of Schlemm patent. |
| No. 301. Sarah H., æt. 75. *R.* came bad 2 years ago while she was in bed with a broken leg. Pain then. At excision, T. 2. Dense leucoma of cornea; no anterior synechia. *L.* T. n. | Extreme atrophy. Only a few nuclei and a little sclerosis tissue remains. Ciliary folds much sclerosed. | Sclerosed, periphery adherent. | Access closed .. |
| No. 81. James D., æt. 35. *R.* failed gradually 3 years; much pain 2 years. *L.* T. n. | Inflamed, moderately atrophied. | Periphery adherent. | Access closed .. |
| No. 303. Wm. F., æt. 80. Glaucoma in *R.* about 12 months. Pain 6 weeks. *L.* also glaucomatous, but less severely so. Iridectomy in both 3 weeks ago. T. in *R.* not relieved. History more fully in vol. ix, p. 395. | Much atrophied, much sclerosis tissue in a rather early stage, and containing a few nuclei. | Limb removed at upper part by iridectomy; periphery free at lower part. | Access open, but some nuclei in lig. pect. and c. of S., which is very small. |
| No. 499. Amos B., æt. 48. *R.* came foggy 4 years ago. Now no p.l. for 3 years. Moderate pain 1½ years, and much inflammation and pain during the last 4 months. At excision T. 2. There is a staphyloma of the inner equator the size of a large pea. The a.c. is of fair depth, except at its margins, where it is obliterated. There are extensive posterior synechiæ. Lens cataractous and *in situ*. | Extremely atrophied, inflamed, early sclerosis. | Much inflamed; enlarged blood-vessels in its base. | Access closed .. |

*accompanied by Retinal Hæmorrhages.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged .. | Moderately deep cup, extending over the entire disc. | Neuritis, also sclerosis, especially of the sheath of the central artery. | Have hyaline walls. | .. .. | .. .. |
| Enlarged .. | Universal cup, not very deep. | Much vascular sclerosis. | A little hypertrophied. Capillaries gape; walls hyaline. | Some arteries of choroid much thickened in neighbourhood of optic disc. Anterior part of retina is very cystic. | Leucoma apparently secondary in time to the glaucoma. |
| Not enlarged. | Cupped .. | Much neuritis, some sclerosis, numerous nuclei in sheath of central artery. | Small blood-vessels of anterior part have walls much thickened and hyaline. | .. .. | .. .. |
| Not enlarged, but long internal ciliary arteries are vastly enlarged and thin-walled. | Shallow shelving cup. | Much vascular sclerosis. | Walls much thickened, especially those of the smaller arteries, which have their lumen nearly obliterated. | .. .. | No note as to condition of kidneys or heart. |
| Moderately enlarged. | Small cup .. | Neuritis and vascular sclerosis. | Walls of arteries hypertrophied, capillaries with aneurysmal dilatations. | Choroid at staphyloma inflamed, thin. Retina corresponding to it is very cystic. | The principal feature about the case is the marked inflammatory condition of the uveal tract and the cystic retina at the area of local staphyloma. |

GROUP 3.—*Eight cases in which a blow or wound attended by comparatively slight which the eye has*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 101. Hy. W., æt. 76. Minute perforating corneal wound 14 days ago. T. 1 at excision. In both eyes M = about 6 D. History more fully reported in vol. ix, p. 396. | Fair size and structure. Some translucent substance between bundles of circular fibres. | Periphery widely free, except at the one point, where prolapsed into internal orifice of the wound. | Wide open, except at the point where plugged by iris. |
| No. 371. Thomas M., æt, 40. Rupture of upper sclero-corneal junction by a blow 21 days before excision. T. 2. History more fully in vol. ix, p. 404. | Much atrophied, much sclerosis. Sclerosis and atrophy considerable, but not so great at lower part of cil. circle. | Gone, perhaps escaped through rupture. | Open .. .. |
| No. 421. James W., æt. 50. Blow on *L.* with fist 21 days ago. P.l. soon went, and severe pain came on lasting till excision. Lens displaced behind iris above, but in a.c. below. T. 3. Usual health good, but appears to have had gout. | Inflamed, early stage of sclerosis; there is some atrophy; thickness = ·52 mm. | Periphery applied .. | Access closed .. |
| No. 214. Joseph S., æt. 56. Blow 14 weeks ago, dislocating lens. T. n. then. Ten weeks ago the lens was removed and vitreous lost by an imperfect iridectomy upwards. Tension then came on. At excision T. 2. History more fully in vol. ix, p. 407. | Some atrophy (·54 mm.), moderate translucent substance among circular fibres. Some inflammation. | Periphery applied above, limb partly gone, stump in scar of iridectomy. Periphery free below. | Inflamed .. .. |
| No. 352. Mary W., æt. 38. Sclerotic perforated behind upper ciliary region by glass 5 months ago. No foreign body in eye. T. 1. History more fully in vol. ix, p. 397. | Upper part, some atrophy, thickness = ·55 mm. Some sclerosis of circular fibre region and of ciliary folds. Lower part better developed, = ·7 mm. Some sclerosis as above. | Periphery free .. | Well open .. .. |

*inflammatory disturbance has been rapidly followed by Glaucoma, on account of been enucleated.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Much enlarged. Walls extremely thin. | Central pit, like a physiological cup. | Neuritis, some translucent substance in sheath of central artery. | Walls seem neither hypertrophied nor hyaline. | Lens dislocated into vitreous chamber, but retains its upper and outer attachments. | Lig. pect. very thin, and considerably extended backwards. |
| Enlarged .. | A little swollen. | Neuritis, some translucent substance round new blood-vessels. | Walls seem neither hypertrophied nor hyaline. | Inflamed patches, in choroid like plastic. | Sclerosis in muscle much in advance of that in O.N. |
| Enlarged .. | Central physiological pit; margins much swollen. | Much neuritis, some translucent substance in sheath of central artery. | Walls hypertrophied. | Numerous hæmorrhages in retina. | The condition of the retinal b. vs. is very interesting in relation to the duration of the disease. |
| About normal. | Not cupped. Nuclei round the blood-vessels of papilla. | Neuritis .. | Nuclei round blood-vessels of retina, otherwise seem normal. | Many spots of disseminated choroiditis in a state of activity. Probably some enlargement of the supra-choroidal lymph-space as a clear gap of considerable size round long internal ciliary artery and nerve. | An exceptional case in many respects. Anterior part of retina cystic. |
| About twice as large above as below, where normal. | A central shallow pit. Sclerosis of sheaths of arteries of papilla. | No neuritis, bands of sclerosis not pressing much on nerve tissue. | Walls neither hypertrophied nor hyaline. | Choroid and Ret. destroyed at seat of wound by cicatricial tissue from sclerotic. Small post. synechiæ. | .. .. |

GROUP 3.—*Eight cases in which a blow or wound attended by comparatively slight which the eye has*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. | |
|---|---|---|---|---|
| No. 437. Joseph P., æt. 31. Blow on *R.* 8 months ago, after which sight faded. Especially painful last 14 days. T. 1. *L.* myopic 4 D. | Atrophied, inflamed, much sclerosed, but not very densely. | Periphery adherent. Pupil small, excluded, and occluded. | Access closed | .. |
| No. 279. Isaac D., æt. 51. Blow on *R.* about 1 year ago, apparently followed by iritis. A second slight accident 11 months ago. P.l. rapidly went; and severe pain on *R.* side of head and in eye. At excision pupil irregular and excluded. A small opacity on cornea. T. 2. | Extreme atrophy and sclerosis. | Periphery adherent. | Access closed | .. |
| No. 218. Thomas B., æt. 58. *L.* dim 4 years. Blow 4 weeks before excision. Remains of soft lens in a.c. Much pain. T. 2. | Atrophied. Some translucent substance just fibrillating. | Periphery adherent. | Access closed | .. |

GROUP 4.—*Twenty-two cases in which Increased*

A. In persons over 20

| | | | | |
|---|---|---|---|---|
| No. 83. Wm. K., æt. 57. *R.* vascular leucoma and staphyloma of cornea with entanglement of iris after perforating corneal ulcer 3 months ago. Now cornea leucomatous and prominent, T. +. | Atrophied; inflamed. Pigment cell layer of p.c.r. scattered and partly destroyed. | Inflamed; applied to posterior corneal surface, periphery adherent. | Access closed | .. |
| No. 496. George K., æt. 47. *L.* corneal ulcer 5 months. Iris applied to cornea, and lens in contact with iris. T. full. | Of fair size, inflamed, early sclerosis. | Somewhat inflamed, periphery applied. | Access closed, inflamed. | |

*inflammatory disturbance has been rapidly followed by Glaucoma, on account of been enucleated.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Enlarged .. | .. | Neuritis, especially on one side; much sclerosis of sheath of central artery. | Retina detached. Walls of blood-vessels as above. | Vitreous quite small, totally detached from posterior part of globe. Lens opaque *in situ*. | .. .. |
| Much enlarged. | Cupped .. | Decided vascular sclerosis. | Walls of blood-vessels as above. | .. .. | .. .. |
| Enlarged. Walls extremely thin. | Cupped .. | Neuritis and vascular sclerosis, apparently older than in muscle. | Walls of blood-vessels as above | .. .. | Glaucoma probably of a longer duration than 1 year. |

*Tension has followed a Perforating Corneal Ulcer.*

years old. Eight cases.

| | | | | | |
|---|---|---|---|---|---|
| Not enlarged. | Not cupped.. | Neuritis and narrow bands of vascular sclerosis. | Walls neither hypertrophied nor hyaline. | .. .. | Herpes frontalis on *R.* side 13 years ago left then a small leucoma of cornea. |
| Not enlarged. | Central pit; early stage of sclerosis of walls of artery of papilla. | Neuritis and very early vascular sclerosis. | Walls neither hypertrophied nor hyaline. | .. .. | .. .. |

GROUP 4.—*Twenty-two cases in which Increased*

A. In persons over 20

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 125. John H., æt. 60. Has had paralysis of *L.* seventh nerve for 12 years. Ulcer of *L.* cornea of 6 months' duration only. Cornea leucomatous and prominent. T. +. | Thin; inflamed clusters of nuclei in the connective tissue internal to muscle. | Adherent; atrophied. | Access closed, inflamed. |
| No. 18. John C., æt. 49. Perforating ulcer of *L.* cornea 7 months ago, said to be from exposure. Now cornea leucomatous and prominent. | Moderate atrophy; inflamed much. | Adherent; atrophied; also inflamed much near ulcer. | Access closed, inflamed. |
| No. 776. Jane R., æt. 60. Says that she had erysipelas of the face 9 months ago. After this got somewhat better; the *R.* eye came bad with a perforating ulcer. Cornea as above. T. 2. | Atrophied, much inflamed, some early sclerosis. | Adherent; atrophied; also inflamed much near ulcer. | Access closed, inflamed. |
| No. 287. Mary H., æt. 60. *L.* staphyloma of leucomatous cornea 1 year. T. +. | A little atrophied and inflamed; much sclerosis of circular fibres. | Adherent; atrophied; also inflamed much near ulcer. | Access closed, inflamed. |
| No. 181. Alfred M., æt. 50. *R.* leucoma of cornea from perforating ulcer 2 years ago. Bulging of thinned translucent upper corneal margin. T. +. | In *upper* ciliary region, total atrophy, some inflammation and much early sclerosis. In *lower;* musc. fair, early sclerosis of circ. fibres. | Base much inflamed, connective tissue uniting iris and cornea. | Access closed, inflamed. |
| No. 16. Wm. G., æt. 51. *L.* lost 2 years ago, by a perforating ulcer occurring, together with herpes frontalis. T. full. *R.* myopia = 3 D. | Moderate atrophy; inflamed. | Adherent and atrophied. | Access closed .. |

*Tension has followed a Perforating Corneal Ulcer.*

years old. Eight cases.

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Slight central depression, margin a little swollen. | Neuritis .. | Walls neither hypertrophied nor hyaline. | .. .. | .. .. |
| Not enlarged. | Mod. deep shelving pit. | Neuritis .. | Walls neither hypertrophied nor hyaline. | .. .. | .. .. |
| Not enlarged. | Mod. deep shelving pit. | Neuritis and early sclerosis. | Somewhat thick as if hypertrophied. | .. .. | .. .. |
| Not enlarged. | Mod. deep pit. | Neuritis and vascular sclerosis. | Somewhat thick as if hypertrophied. | .. .. | .. .. |
| Not enlarged. | Mod. deep pit. | Much neuritis and vascular sclerosis. | Walls neither hypertrophied nor hyaline. | .. .. | .. .. |
| Not enlarged. | Shallow depression. | Neuritis and early sclerosis. | Walls neither hypertrophied nor hyaline. | Fibres of intersheath space of nerve much hypertrophied. | Patient myopic. Herpes frontalis. |

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 463. Samuel L., æt. 4. *L.* staphyloma of leucomatous cornea from perforating ulcer 6 months ago. T. 1. | Not atrophied, inflamed. | One limb entangled in ulcer, atrophied and adherent; opposite limb inflamed, periphery adherent. | Access closed, inflamed, lumen very small. |
| No. 52. Henry M., æt. 7½ months. *R.* staphyloma of leucomatous thickened cornea from corneal ulcer, the result of ophthalmia neonatorum. T. +. | Much inflamed .. | Adherent, in places atrophied; much inflamed near ulcer. | Access closed, inflamed, lumen very small. |
| No. 428. Henry F., æt. 4. *L.* staphyloma corneæ from perforating ulcer 8 months ago. T. +. | Much inflamed .. | Adherent, in places atrophied, much inflamed near ulcer. Connective tissue on posterior surface of iris and of ciliary body. | Access closed, inflamed, lumen very small. |
| No. 146. William S., æt. 5. *L.* staphyloma corneæ from perforating ulcer 1 year ago. T. +. | Much inflamed .. | Adherent, atrophied, but in places inflamed. | Access closed, inflamed, lumen very small. |
| No. 363. George C., æt. 14. *R.* staphyloma corneæ from perforating ulcer 1½ years ago. T. +. *L.* myopia = 7 D. | Atrophied, inflamed, perhaps a little sclerosed. | Adherent, atrophied, but in places inflamed. | Access closed, inflamed, lumen very small. |
| No. 30. Joseph R., æt. 16. *L.* staphyloma corneæ from burn with molten lead 2 years ago. | Inflamed and a little atrophied. | Adherent and atrophied. | Access closed, inflamed, lumen very small. |

*Tension has followed a Perforating Corneal Ulcer.*

years old. Fourteen cases.

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Central funnel-shaped pit. | Neuritis and early vascular sclerosis. | Walls neither hypertrophied nor hyaline. | Choroid contains occasional clusters of nuclei in its outer layers, and a general diffused increase of nuclei besides. | .. .. |
| Not enlarged. | Central funnel-shaped pit. | Much neuritis. | Walls neither hypertrophied nor hyaline. | Choroid has a general diffused increase of nuclei, its blood-vessels remain full of corpuscles. | .. .. |
| A trifle large. | Central funnel-shaped pit. | Neuritis and early vascular sclerosis. | Walls perhaps somewhat hypertrophied. Some episcleritis. | Choroid has a general diffused increase of nuclei, its blood-vessels remain full of corpuscles. | .. .. |
| Not enlarged. | .. .. | Neuritis .. | Walls neither hypertrophied nor hyaline. | Choroid has general diffused increase of nuclei, and blood-vessels full of corpuscles. | .. .. |
| Enlarged .. | Central deep pit; dense sclerosis of walls of blood-vessels of papilla. | Neuritis and vascular sclerosis. | Walls hypertrophied. | Choroid has general diffused increase of nuclei, and blood-vessels full of corpuscles. A layer containing irregular scattered pigment exists on the internal surface of ciliary body. | .. .. |
| Not enlarged. | (?) | Neuritis .. | Walls neither hypertrophied nor hyaline. | Choroid has general diffused increase of nuclei. | .. .. |

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 376. Keziah M., æt. 15. *R.* staphyloma after corneal ulcer 4 years ago. T. 3 before an iridectomy was done recently. At excision, T. n. Globe removed on account of pain. | Inflamed, perhaps a little sclerosis of region of circular fibres. | *One limb* in ulcer, *other* inflamed, periphery adherent and atrophied. | Access closed, inflamed, lumen very small. |
| No. 175. Elizabeth F., æt. 16. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +. | Somewhat inflamed, a little atrophied. | Adherent, atrophied mostly, but inflamed in places. | Access closed, inflamed, lumen very small. |
| No. 109. Arthur L., æt. 12. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +. | Much inflamed, stretched, and atrophied; thin irregular layer containing pigment on the internal surface of the ciliary body. | Adherent and atrophied. | Access closed, inflamed, lumen very small. |
| No. 347. Thomas R., æt. 12. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +. | *Upper ciliary region*, extreme atrophy; some inflammation; no sclerosis. *Lower*, inflamed; mod. atrophy; abundant connective layer, containing pigment on inner surface of ciliary body. | Adherent and atrophied. | Access closed, inflamed, lumen very small. |
| No. 34. Sarah W., æt. 13. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. n. now. | Moderately atrophied and much inflamed; a thin layer of newly formed nuclear bodies on internal surface of ciliary body. | Mostly adherent and atrophied, especially at base; limb where free inflamed. | Access closed, inflamed, lumen very small. |

*Tension has followed a Perforating Corneal Ulcer.*

years old. Fourteen cases.

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Perhaps a trifle enlarged; walls of good thickness. | Central pit, edges swollen. | Neuritis (?) (nerve very short in specimen.) | Walls neither hypertrophied nor hyaline. | Choroid has general diffused increase of nuclei. | .. .. |
| Not enlarged. | Cupped .. | Neuritis and vascular sclerosis; numerous new blood-vessels. | Walls neither hypertrophied nor hyaline. | Choroid has general diffused increase of nuclei. | Anterior part of retina is very cystic. |
| Not enlarged. | Cupped .. | Neuritis and vascular sclerosis; numerous new blood-vessels. | Walls neither hypertrophied nor hyaline. | Choroid has general diffused increase of nuclei, and is œdematous. | .. .. |
| Somewhat enlarged, but walls of fair thickness. | Central pit. lam. crib. very concave; concavity nearly filled by nuclear tissue; margins of papilla swollen. | Dense vascular sclerosis. | Walls hypertrophied; lumen very small, or wanting; black pigment often in or round their wall. | Abundant nuclei in ch. and in adherent retina. Pigment epithelium layer destroyed, its pigment black and irregularly scattered. | Suspicion of hereditary syphilitic taint. |
| Not enlarged. | Fairly deep, universal cup. | Much neuritis. | Walls neither hypertrophied nor hyaline. | .. .. | .. .. |

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 38. Edward P., æt. 14. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. formerly increased, now — T. 3. | Inflamed, and containing between its bundles of fibres numerous red blood corpuscles, no sclerosis; layer on ciliary body, as above. | Mostly adherent and atrophied, especially at base; limb where free inflamed. | Access closed, inflamed, lumen very small. |
| No. 468. Rebecca R., æt. 16½. *R.* cornea flat, leucomatous. Globe enlarged in antero-posterior and lateral diameters, and T. + from corneal ulcer when 7 weeks old. Some nystagmus of *both*. *L.* myopic about 7·5 D. | Atrophied and much inflamed; layer on ciliary body, as above. | Mostly adherent and atrophied, especially at base; limb where free inflamed. | Access closed, inflamed, lumen very small. |
| No. 224. Emma M., æt. 19. Dense leucoma of cornea from ulcer in infancy. Ciliary region of sclerotic, and cornea thin and staphylomatous. Globe enlarged. | Almost total atrophy, some inflammation; connective layer on internal surface of ciliary body. | Adherent and extremely atrophied. | Access closed, inflamed, lumen very small. |

GROUP 5.—*Five cases in which the anterior part of the Globe is especially enlarged Traumatic or Ulcerative Origin, and whether with*

| | | | |
|---|---|---|---|
| No. 123. Mary H., æt. 7½. Congenital buphthalmos of both eyes. T. 2. Iridectomy attempted, followed by hæmorrhage into cavity of vitreous and under choroid. Immediate excision. | Atrophied; inflamed, fibres separated by much hæmorrhage. | Atrophied, and adherent to cornea. | Closed .. .. |

*Tension has followed a Perforating Corneal Ulcer.*

years old. Fourteen cases.

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Fairly deep, universal cup. | Much neuritis. | Walls neither hypertrophied nor hyaline. | Choroid has diffused nuclear increase; blood-vessels full. | .. .. |
| Not enlarged. | Not cupped, appearances characteristic of myopia. | A little neuritis; some translucent substance round smaller blood-vessels; numerous vessels of new formation in walls of central artery. | Walls neither hypertrophied nor hyaline. | Choroid contains patches of dense nuclear bodies in neighbourhood of macula. A little episcleritis. | Globe larger in lateral than in vertical diameter. |
| Not enlarged. | Deep, rounded cup. | Neuritis and much sclerosis. | Walls neither hypertrophied nor hyaline. | Choroid has diffused nuclear increase, and blood-vessels full. | .. .. |

*in consequence of Increased Tension from Disease occurring in Infancy, whether of Anterior Synechia or with deep Anterior Chamber.*

| | | | | | |
|---|---|---|---|---|---|
| Not enlarged. | Central pit .. | Neuritis and vascular sclerosis. | Walls of capillaries of int. granular layer much thickened and hyaline. | Ch. contains diffused nuclear increase, and full blood-vessels. It is thickened. | Patient is in all probability syphilitic. |

GROUP 5.—*Five cases in which the anterior part of the Globe is especially enlarged Traumatic or Ulcerative Origin, and whether with*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 69. Constant C., æt. 12. Buphthalmos from penetrating wound 8 years ago. + T. | Much inflamed and somewhat atrophied. | Atrophied, and adherent to cornea. | Closed .. .. |
| No. 787. Arthur M., æt. 10. Buphthalmos from blow in infancy. Pupil occluded and excluded. Universal post. synechiæ. A.c. extremely deep. | Much inflamed .. | Universal posterior synechia. | (?) |
| No. 599. Ann L., æt. 22. Buphthalmos and marginal corneal staphyloma above from wound when æt. 5. + T. at excision. | Atrophied, more especially above, and moderately inflamed. | Atrophied, and adherent to cornea. | Access closed .. |
| No. 760. John A., æt. 54. *R.* buphthalmos from ulcer when æt. 1. T. n. now. *L.* V = $\frac{20}{20}$. | *Above,* atrophied much, and slightly inflamed. *Below,* much inflammation and atrophy. | Atrophied, and adherent to cornea. | Access closed .. |

*in consequence of Increased Tension from Disease occurring in Infancy, whether of Anterior Synechia or with deep Anterior Chamber.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Deep central cup. | Much neuritis and early vascular sclerosis. | Walls of arteries much thickened. | Ch. contains diffused nuclear increase, and full blood-vessels. It is thickened. | .. .. |
| A little enlarged. | Retina detached. | Neuritis and some vascular sclerosis. | Walls neither hypertrophied nor hyaline. | Choroid near o. d. contains considerable excess of nuclear bodies, but its innermost stratum is apparently converted into connective tissue. | .. .. |
| Enlarged .. | Deep cup .. | Neuritis and decided vascular sclerosis. | Apparently thickened. | Ch. contains diffuse nuclear increase. | .. .. |
| Enlarged .. | Shallow shelving cup. | Neuritis and much vascular sclerosis. Much sclerosis of walls of arteries of papilla, nearly obliterating their lumen. | Walls of arteries immensely thickened and translucent, containing a few nuclei. In other places, where there is abundant choroido - retinitis, the pig. epith. is destroyed, the pig. black and irregularly scattered. Some is found round the blood-vessels of which the lumen is sometimes obliterated. | .. .. | .. .. |

GROUP 6.—*Three cases of Glaucoma*

| History. | Ciliary muscle. | Iris. | Canal of Schlemm. |
|---|---|---|---|
| No. 509. James F., æt. 42. *L.* sight very dim 3 months ago. Now it presents distinct iritis serosa, not at all severe. *R.* a *little* dim 3 months ago. Iridectomy was done on *this* eye then. *R.* at *excision;* much swelling at operation wound. T. 2. Dots on posterior corneal surface, &c. | Not atrophied, much inflamed. | Periphery free, except at operation wound. Iris is much inflamed. | Much inflamed. Access not closed by iris, except near operation wound. |
| No. 285. Sarah F. *L.*, cataract 1 year. Inflamed 6 months. T. +. Iridectomy 5 months. At *excision L.* much lymph in pupil. *R.* operated on for cataract 1½ years ago. Now moderate vision. | Not atrophied, much inflamed. Apparently some sclerosis of internal part of ciliary body. | Periphery widely free. | Inflamed .. .. |
| No. 274. Elizabeth H. *R.* dim 3 years. Now T. +. Appearances typical of what is known as iritis serosa. *L.* about normal. | Not atrophied, much inflamed, no sclerosis. | Periphery widely free. | Inflamed .. .. |

*due to "Iritis Serosa," &c.*

| Blood-vessels of ciliary region. | Optic disc. | Optic nerve. | Retinal blood-vessels. | Other pathological conditions. | Remarks. |
|---|---|---|---|---|---|
| Not enlarged. | Central pit .. | Considerable neuritis. | .. .. | Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed. | The iridectomy was not done at Moorfields Hospital. |
| Not enlarged. | Not cupped . | Some neuritis. | .. .. | Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed. | .. .. |
| Not enlarged. | Not cupped . | Some neuritis. | .. .. | Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed. | .. .. |

## EXPLANATION OF FIGURES IN CURATORS' PATHOLOGICAL REPORT.

### PLATE I.

FIG. 1.—Female, æt. 40. To illustrate the normal anatomy of the parts. Death from blood-poisoning.

FIG. 2.—Case 274 in Curator's Report (see page 128). Increased tension probably of long standing. Four months under observation in the Out-patient's Department.

FIG. 3.—Case 101 (see page 114). Increased tension of less than 14 days' duration.

FIG. 4.—Case 483 (see page 106).

### PLATE II.

FIG. 5.—Case 401 (see page 96).

FIG. 6.—Case 452 (see page 104). In this case there were marked inflammatory symptoms, and the aqueous chamber at its centre was deeper than normal.

FIG. 7.—Case 395 (see page 94).

Fig. 8.—Case 338 in Curator's Register. A young man, æt. 19, had been when two years old the subject of a perforated corneal ulcer. The eye became apparently the subject of some secondary increase of tension. *At excision*, it is enlarged especially in an antero-posterior direction, and its posterior segment is the subject of a staphylomatous thinning. There is slight increase of connective tissue in the nerve, but nothing comparable with that seen in the next figure.

FIG. 9.—Case 340 (see page 102). It shows in transverse section the typical condition of optic nerve found in the glaucoma of adults and old persons, described in the Report as Vascular Sclerosis.

Fig 1. ×15.

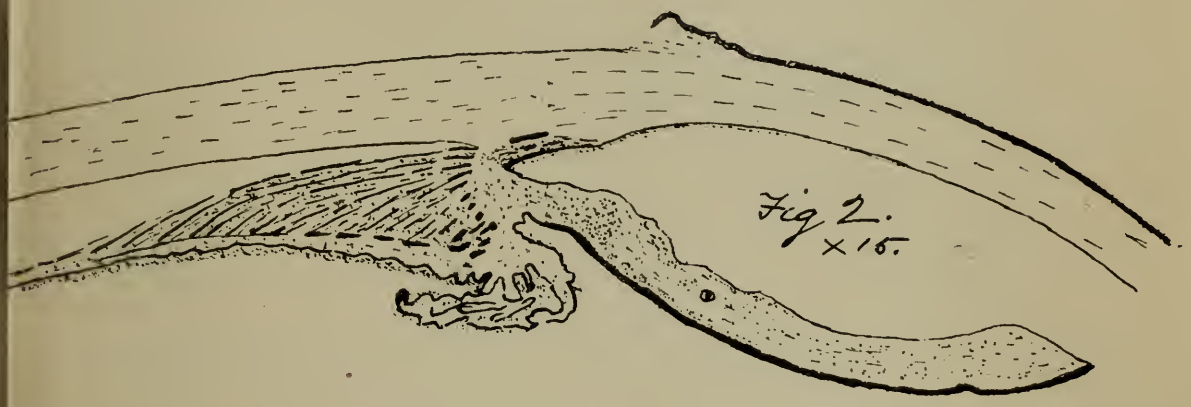

Fig 2. ×15.

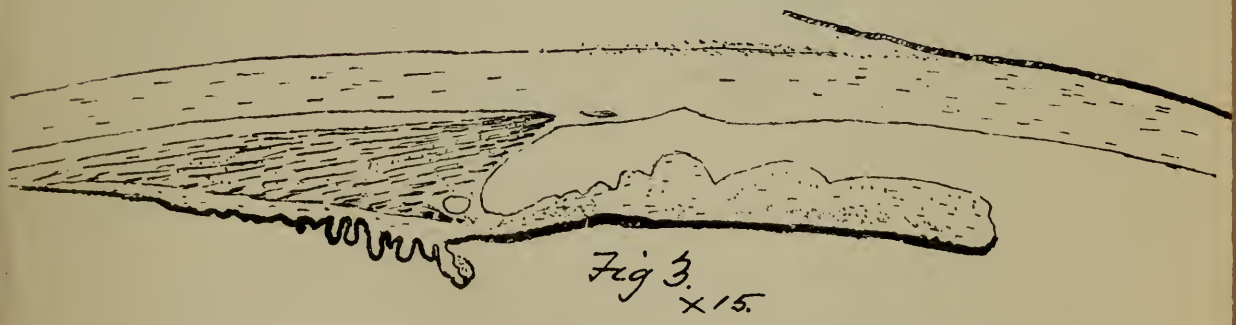

Fig 3. ×15.

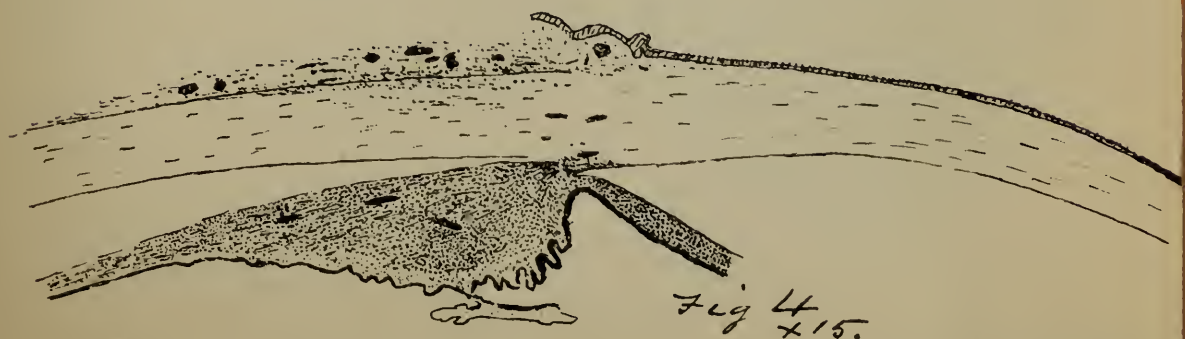

Fig 4. ×15.

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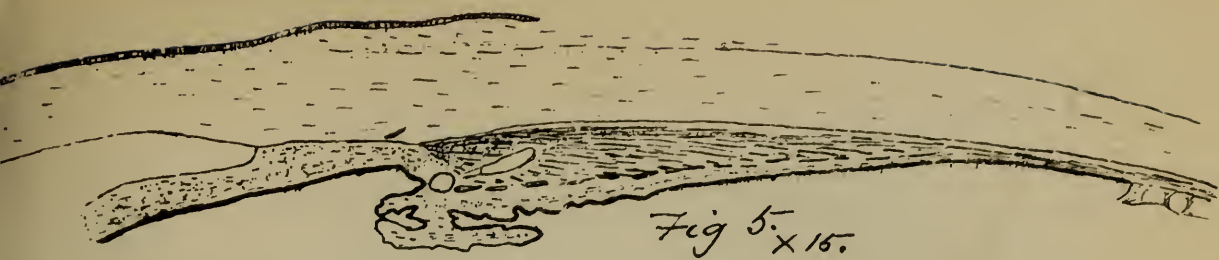

Fig 5. x 15.

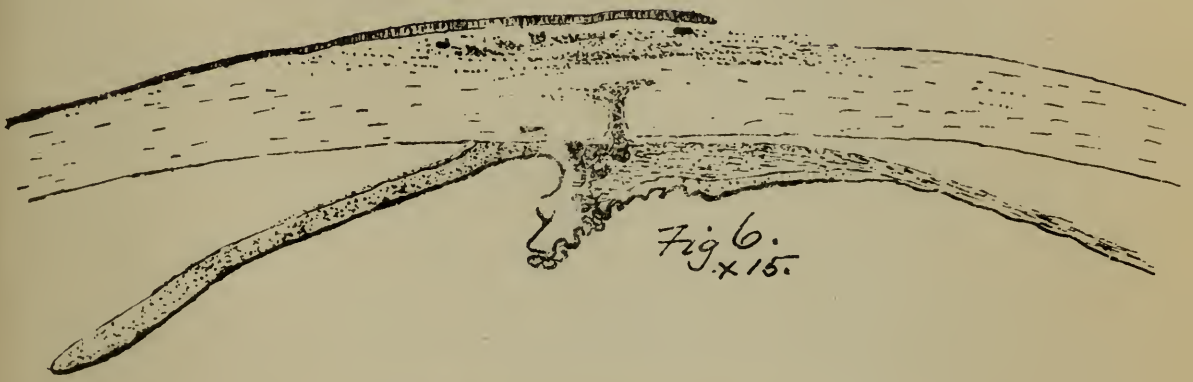

Fig 6. x 15.

Fig 7. x 15.

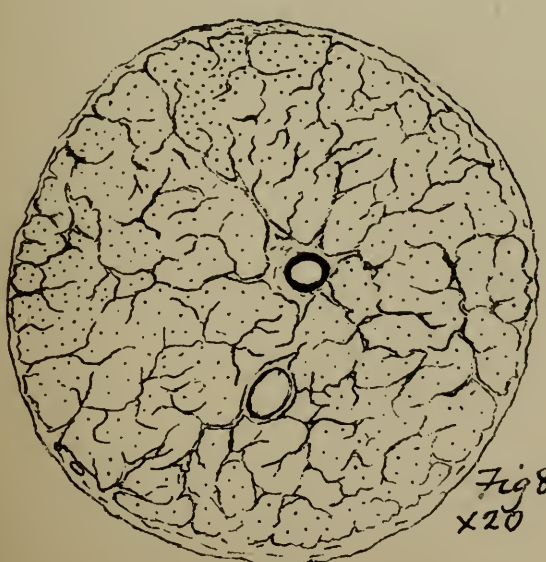

Fig 8 x 20

Fig 9. x 20.

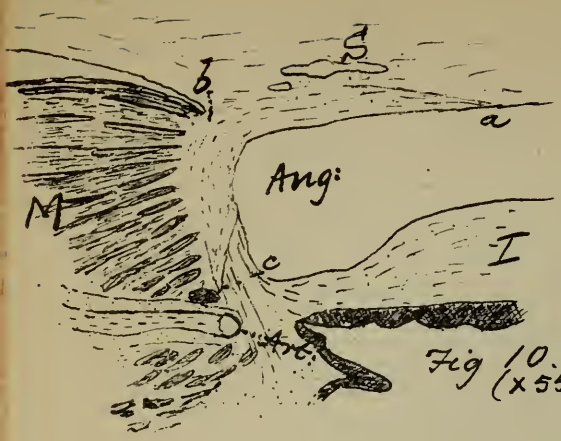


Fig 10. (×55)

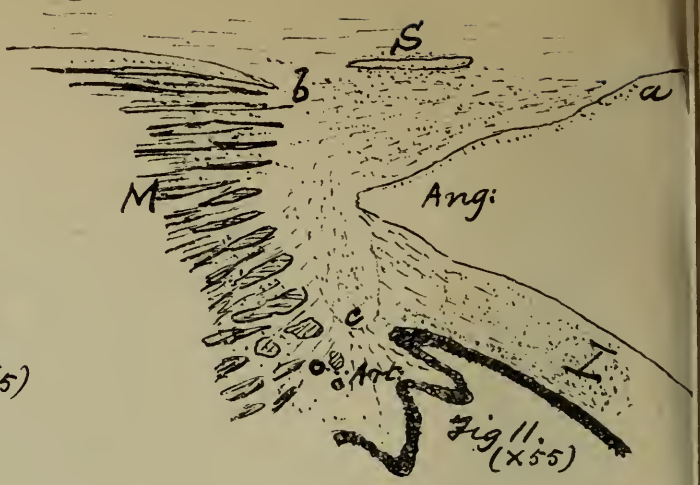


Fig 11. (×55)

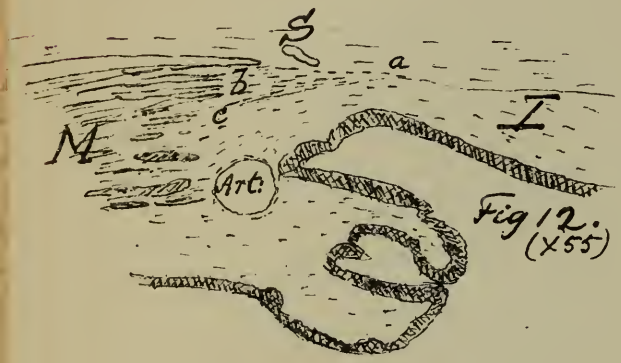


Fig 12. (×55)

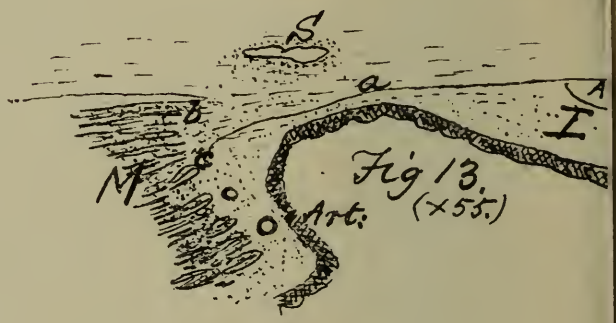


Fig 13. (×55.)

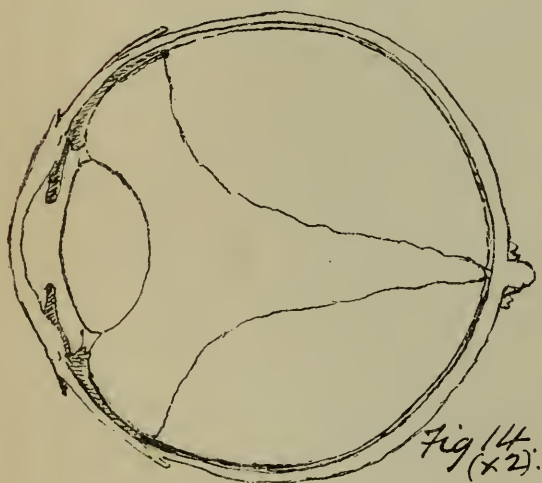

Fig 14. (×2).

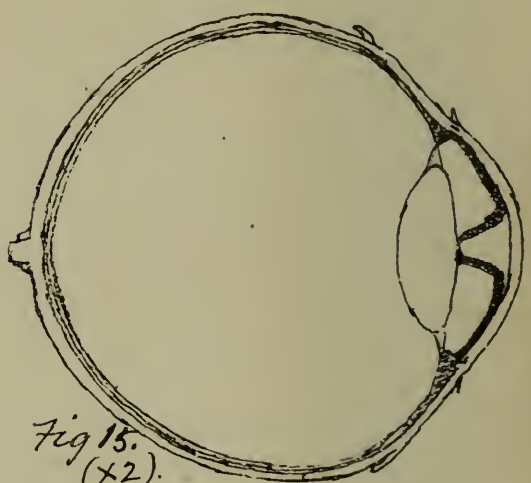

Fig 15. (×2).

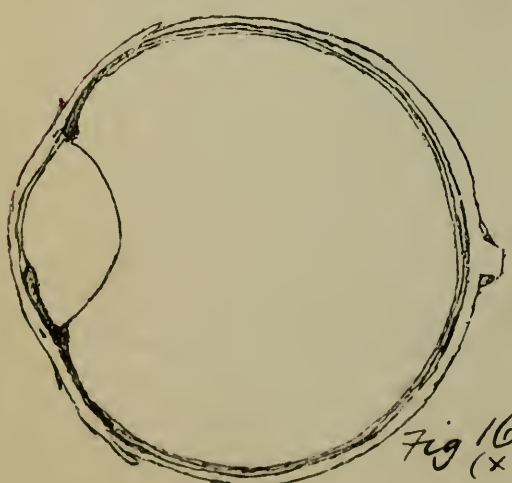

Fig 16. (×2).

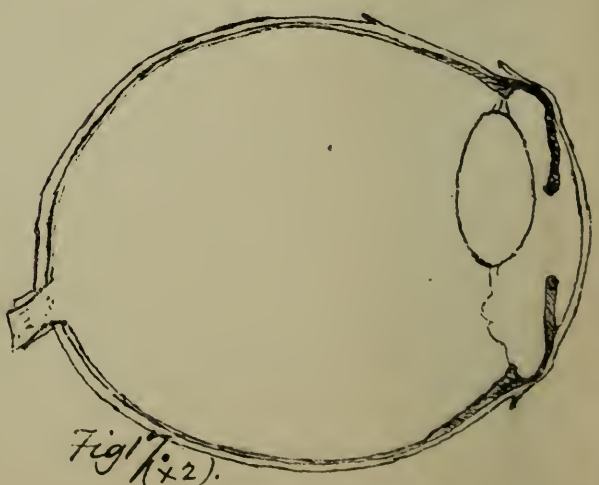

Fig 17. (×2).

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PLATE III.—Explanation of diagrams to show the change in shape of the Ligamentum Pectinatum, which is the principal cause of the approximation of the periphery of the iris to the cornea in glaucoma.

The part specially affected is that named by Gerlach the Ligamentum Annulare (see Gerlach "Beiträge zur normalen Anatomie des Menschlichen Auges." Leipzig, 1880). The most anterior stratum of this by its connexion on the one hand with Descemet's membrane, and on the other with the connective tissue framework of the iris narrows the peripheral part of the anterior chamber of the Aqueous. The more posterior and thicker stratum by *its* diminution in size contracts together the fibres of origin of the meridional part of the ciliary muscle. The terms here used are adopted from Gerlach :—

*a* = inner angle.
*b* = outer angle (usually rounded).
*c* = posterior angle (usually acute).
*bc* = outer border.
I. = Iris.
S. = Canal of Schlemm.
Art. = Circulus Arteriosus.
M. = Meridional part of ciliary muscle.
Ang. = Angle of Anterior Chamber of Aqueous.

FIGS. 10, 11, 12, and 13, are enlarged representations of the Ligamenta Pectinata of Figs. 1, 2, 5 (reversed), and 6 respectively.

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## APPENDIX TO CURATOR'S REPORT.

Since the above report has been in the press four eyes with increased tension have been examined immediately after excision after being frozen.

The eyes were divided vertically in an antero-posterior plane, cutting from above downwards.

The sections are represented in the accompanying figures.

FIG. 14.—John McD., æt 57. 30 years ago he had an accident with gunpowder, after which black particles remained imbedded in his left cornea. 12 months ago he could still read large print, though his eye had become considerably myopic. Three weeks ago it became painful and a little red : a few days later he accidentally discovered it had only p.l. At excision T 1½. Measurements of *globe :* Antero-posterior 25 mm., vertical 23·5 mm. *Lens* diameter 9·3 mm. Antero-posterior 4·75 mm. Depth of a.c. from posterior corneal surface 2 mm.

FIG. 15.—Thomas I., æt. 19. Vision lost by iritis three years ago. At excision T 2. Pupil excluded and occluded. *Globe :* Antero-posterior 25·5 mm., vert. 24·75 mm. *Lens* diam. 8·75. Antero-posterior 2·75 mm.

FIG. 16.—James Buckland, æt. 59. Seven months ago his eye became inflamed and painful, with small corneal infiltrations and hypopyon ; the pupil was inactive with posterior synechiæ. T + : V = p. l. One month later iridectomy upwards was done. In another month the extensively infiltrated cornea was incised by Sæmisch's method ; since then severe pain till excision. The a.c. is said to have reformed previous to excision. T +. *Globe:* Antero-posterior 25 mm., vert. 24 mm. *Lens* diam. 11 mm. Antero-posterior 5·5 mm. Abundant soft material immediately inside lens capsule.

FIG. 17.—Thomas C., æt. 3. Glioma with increased tension. The posterior segment of the globe full of glioma matter, but the aqueous chamber is as yet quite free. It is much increased in size.

## ON SOME CHANGES WHICH OCCUR IN THE BLOOD-VESSELS IN DISEASES OF THE EYE, CONSIDERED IN THEIR RELATION TO GENERAL PATHOLOGY.

By Walter Edmunds, M.D., and W. A. Brailey, M.D.

The structure of the eye in comparison with its size is very complex; in it are found representatives of the chief tissues of the body; and as the eye, in addition to numerous local affections, is frequently involved in general diseases, a collection of diseased eyes will afford a rich field for pathological study.

Such a collection there is in the museum of Moorfields Hospital, where microscopical preparations of a very large number of eyes are preserved, the specimens being particularly easy of comparison from their having all been made in the same manner, namely, hardened in chromates, stained in log-wood, and mounted in Dammar. The following observations are based on the study of this collection.

The thickening of the coats of the arteries which occurs throughout the body in some form of Bright's disease was pointed out many years ago by Dr. George Johnson, and the discussion which subsequently arose between him, Sir W. Gull, and Dr. Sutton, as to the exact nature and interpretation of the change, again directed attention to this hypertrophy, as to the existence of which there is no question whatever.

In Syphilis, too, hypertrophy of the walls of arteries has been described by Heubner in Germany, and in this country specimens of the disease have from time to time been exhibited at the Pathological Society, more especially when the memorable discussion on Syphilis was being held.*

Widespread arterial change has also been described in

* *See* Pathological Society's Transactions for 1877, where also will be found an account of Dr. Klein's "Observations on the Arterial Changes in Scarlet and Typhoid Fevers."

Diabetes, and an interesting example of this will be found fully related in the last volume of these Reports by Dr. Stephen Mackenzie.

It must not, however, be inferred from these observations that arterial hypertrophy is necessarily a general or widespread disease: for, as we shall endeavour to show, it sometimes is found in the eye alone: the thickened arteries, which have often been described in Retinitis Albuminurica, are part no doubt of a general disease; but a similar condition of vessel is found in Glaucoma, which, at all events in the cases in which it is secondary to injuries, appears to be a purely local affection.

Before describing the changes the blood-vessels undergo in Glaucoma, it is necessary to point out that under this term we include all cases in which the intraocular tension is increased, whether primarily, as in Glaucoma strictly so called, or secondarily, as in Glaucoma supervening on injury, corneal ulcer, or inflammation of the uveal tract. Under this head, too, we include the hæmorrhagic Glaucomas, and the condition known as Buphthalmos, the peculiar appearance to which this name is applied being due merely to the action of increased tension in children in whom the cornea, where not leucomatous, readily stretches.

In another part of these Reports will be found analyses of the morbid appearances in 88 cases of Glaucoma (see page 94): and among the 23 cases of the non-hæmorrhagic form in which the retina was carefully examined, the retinal arteries were distinctly hypertrophied in eight. The proportion is very different in the hæmorrhagic form of the disease (Group II), for out of the 17 cases in which the retina was examined, the arteries were found thickened in all but one. This is not due to any radical difference in the nature of the two forms of the disease, but merely to the fact that hæmorrhages only occur in the more severe or advanced cases, and that consequently only these are included in the hæmorrhagic category.

Group III is composed of cases of comparatively slight

injury to the eye in which it has become necessary to perform excision within a year on account of Secondary Glaucoma. Among these the retinal arteries were only found to be hypertrophied in one case, the history of which is of special interest.

A man, 50 years of age, received a blow on the eye with the fist; he had severe pain almost immediately, with tension becoming greatly increased (T. 3) for three weeks when the globe was excised. On examining the eye, there were found amongst other changes numerous capillary hæmorrhages in the retina, the arteries of which were distinctly hypertrophied. The patient was not the subject of any general disease, nor is there any reason to think that the eye was in any way abnormal before the injury, and if this were the case, the hypertrophy of the arteries must have come on in 21 days.

Among the 22 cases of Glaucoma following perforating ulcers (Group IV), thickened retinal arteries were found in five cases.

Lastly, among the five cases of Buphthalmos (Group V), the same change was seen in three, in one of which the diseased condition of blood-vessels was very manifest, for, while the operation of iridectomy was being performed, the whole globe filled with blood, and it became necessary to excise forthwith.

Thus we see that in a considerable proportion of cases of Glaucoma, the retinal arteries are hypertrophied. The thickened vessels in a later stage contract, and their lumen becomes smaller: in two cases they were impervious, and reduced to the condition of fibrous cords: it may be added that in Glaucoma the vessels never present any proliferation of their endothelium.

In cases in which there has been choroido-retinitis the arteries and their sheaths are frequently found impregnated with pigment molecules, which have apparently come from the diseased pigment epithelium layer of the retina.

The pigment also surrounds the aneurysmal dilatations which these arteries frequently exhibit.*

But the arteries are not the only vessels which are affected: the capillaries often have their walls thickened and composed of a structureless, highly refracting substance, which we have called "hyaline;" they gape on section, and are often aneurysmal. This last condition may be seen either in section in which a capillary happens to be exposed in its length, or by looking at a portion of the retina on the flat.† The retinal veins are not nearly so conspicuously, if at all, affected.

The central artery of the optic nerve is also altered, but the changes it undergoes are of a different character.

In all cases of Glaucoma the optic nerve is found inflamed or sclerosed; and in this inflammation (for sclerosis is probably only a late stage of neuritis) the central artery participates as it passes through the nerve. The only change noticed in the earliest cases is an enlargement of its vasa-vasorum and the appearance of nucleated bodies in its lymph-sheath, that is to say, in the loose tissue which forms part of its adventitia. In the next stage a structureless substance, in which nuclei lie sparsely imbedded, is seen in the lymph-space; and in the later stage this substance becomes fibrillated, and finally converted into a dense fibrous tissue in which no nuclei can be detected.

Some evidence that this is the order in which the changes occur is obtained from a comparison of the duration of the Glaucoma in the cases of Group III.

The last two stages (the translucent and the fibrous) we conceive to be the conditions of artery which Gull and Sutton have included under the name "hyalin-fibroid."

Identical changes have been described in the arteries of

* *See* Poncet (de Cluny), Atlas, pl. LI (1879).

† Retinal aneurisms were first described by Liouville: he found them occurring as part of a general condition which he considered as an aneurysmal diathesis; no increase of ocular tension is mentioned in any of his cases. (Comptes rendus de la Société de Biologie, 1868.)

sclerosed portions of the brain and spinal cord (as we ascertained subsequently to observing them) by Charcot.*

As to the causation of these vascular changes the starting point seems to be the inflammation (or sclerosis) which, as we have said, is found in all cases of Glaucoma. This inflammation affects the central artery in its course through the optic nerve, and thus occasions the changes in its lymph-sheath: the inflammation is propagated forwards to the retinal arteries, but in them it cannot take quite the same form, for they have not (as far as our observations go) lymph-sheaths.

In the retinal arteries it is difficult to define the exact seat of origin of the inflammatory changes, but in the central artery of the nerve it is the muscular coat which appears most often involved.

The varicose condition of the capillaries might, perhaps, be explained by the fact that the thickened and probably inelastic walls of the retinal arteries would more directly transmit the pressure of the blood, which might then be excessive for the altered walls of the capillaries.

If the disease of the retinal arteries is due to the central artery being affected in its course through the optic nerve, it will follow that the choroidal arteries, which have an entirely distinct origin, will not be affected, and, in fact, they are not, at least in the great majority of cases. Indeed, in nearly all the specimens the contrast between the thickened retinal arteries and the natural choroidal vessels is very marked, and quite confirms our proposition that the arterial change is strictly local.

One other vascular change in Glaucoma still remains to be mentioned: the arteries in the ciliary region are often greatly dilated. This change may be well seen in the "Circulus arteriosus iridis major," which is frequently found vastly enlarged, with it walls even more than proportionately thinned.

Thus we see that in Glaucoma there are frequently found in the same eye hypertrophied retinal arteries, those of the

* "Leçons sur les Maladies du Système Nerveux," pp. 181, 1872.

choroid unaltered, and the arteries of the ciliary region dilated and thinned; a condition which is not to be explained by any general arterial disease.

In syphilis the retinal arteries are affected, but in a different manner. Their walls on transverse section are seen dissociated, and thicker than natural, the thickening being more than can be accounted for by the dissociation of the fibres of which they are composed. That this change is probably in the first place inflammatory may be inferred from the frequent presence of staining nuclei in the dissociated walls. It may be a later stage of the syphilitic arteries described by Barlow and others at the Pathological Society. Their appearances closely resemble those observed by Heubner in the intima, and figured in his mongraph (1874).

As to the central artery of the optic nerve, although no doubt its sheath can participate in the results of surrounding inflammation, yet this vessel appears to be unaffected when syphilis is uncomplicated by any other diseased condition.

In syphilis, as in Glaucoma, a varicose condition of the capillaries is sometimes found: also, when the retina is implicated in a choroido-retinitis, the retinal arteries may become pigmented.

In syphilis the choroidal arteries do not appear to be affected, and those of the retina present the same characters, whether the disease be acquired or inherited.

In discussing the distinctly inflammatory diseases of the eye it will be convenient to consider more especially the serous inflammation of the uveal tract, as in this the changes are slower and can be more readily determined.

In this there are two distinct types of arterial disease: one may be observed in the choroid, the other in the retina, which is always affected secondarily and to a less degree.

The choroidal arteries participate in the general character of the choroidal inflammation, which is essentially a proliferation of the fixed corpuscular elements of its stroma.

In the more advanced forms of this inflammation the endothelium of the arteries participates extensively, whereas

the wall presents comparatively but few corpuscular elements. In the iris the smallest arteries are often seen completely blocked by this endothelial proliferation, while the thick, nearly pellucid walls, which characterise them in this structure, are nearly free from staining elements, though they are closely surrounded by the crowded corpuscular elements of the inflamed part; but in the retina, which is always involved to a minor degree in the inflammation, the change is not one of connective-tissue proliferation, but chiefly of diapedesis.

Transverse sections of the retinal arteries show their walls crowded by leucocytes, and thus greatly thickened; their lumen is reduced by a narrow chink, not by endothelial increase, but by the swelling of the thickened walls. On the vitreous surface of the hyaloid, corresponding to the retinal arteries, are leucocytes which have migrated from these vessels.

In the same way it is the connective tissue of the optic nerve which is chiefly affected, for the nuclear bodies which are found in it lie not so much in the loose tissue of the lymph-sheath of the artery as in the dense connective tissue external to it, which is continuous on its outer aspect with the fibrous trabeculæ of the nerve. In conformity with this distribution the nuclei are seen extending horizontally in the framework of the nerve for some little distance. In cases of descending neuritis, on the other hand (the choked disc of authors), we have found leucocytes crowding the loose tissue of the sheath of the artery, the fibrous stroma of the nerve being comparatively free from them.

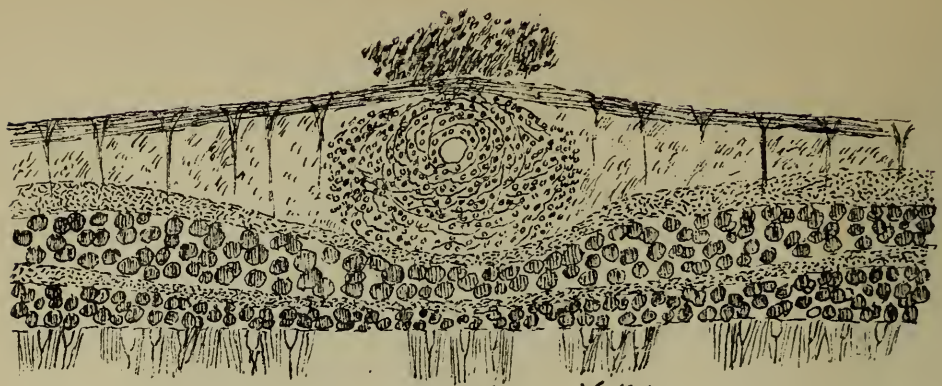

Fig A X 150

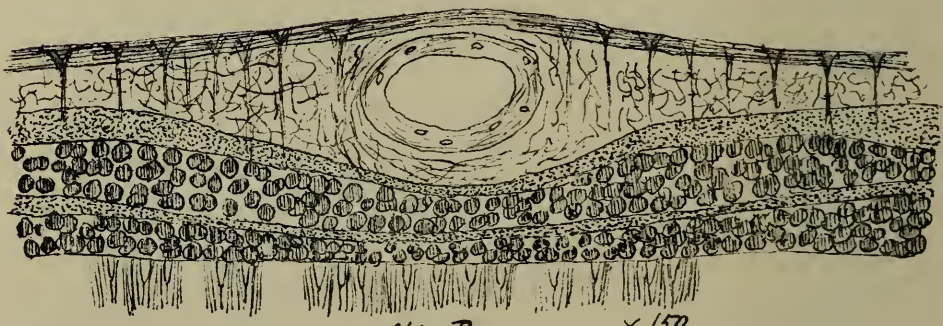

Fig B x 150

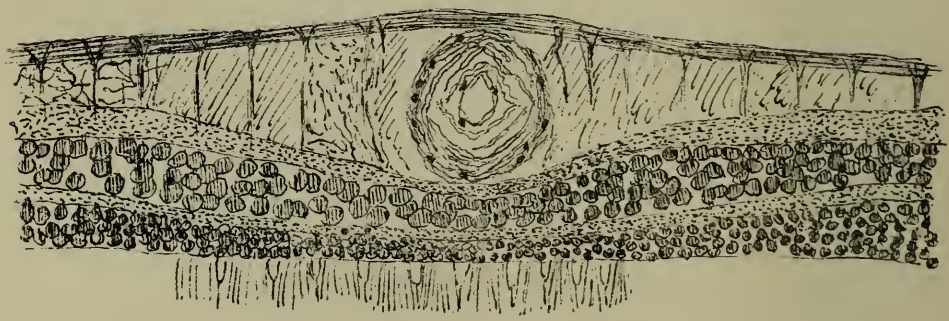

Fig C x 150

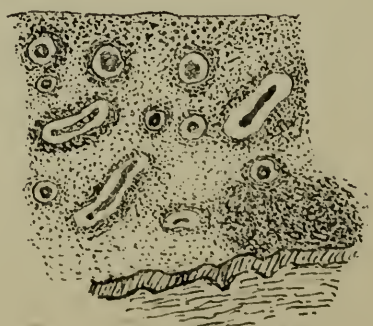

Fig D x 20

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## EXPLANATION OF DIAGRAMS.

FIG. A is the Retina in Case 274, mentioned in the Curator's Report, Group 6. It was a typical case of Iritis Serosa; the eye had been failing for three years.

FIG. B. A typical case of Hæmorrhagic Glaucoma, No. 500 in Curator's Register.

FIG. C. Retinal Artery in a case of Hereditary Syphilis, No. 187 in Curator's Register. This case will be found reported in Mr. Hutchinson's work on Syphilitic Diseases of the Eye and Ear, p. 179.

FIG. D. To show condition of Blood-vessels of Iris in a case of sympathetic inflammation, No. 396 in Curator's Register.

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PART I.

## Original Contributions.

### ON THE TREATMENT OF SYMPATHETIC OPHTHALMIA.

By GEORGE CRITCHETT, F.R.C.S.,

*Consulting Surgeon to the Royal London Ophthalmic Hospital, late Ophthalmic Surgeon to the Middlesex Hospital.*

FEW subjects have, in these latter days, attracted more attention, or excited more painful interest amongst ophthalmic surgeons than sympathetic ophthalmia; nor have we far to seek in explanation of this very natural feeling, for, in the presence of this dread disease, our art is but too often impotent and ineffectual. Athough there exists a sad uniformity of opinion as to the intractable nature of the malady, and as to the frequency, and well-nigh constancy, with which it either seriously impairs, or absolutely destroys the sight; and although there is a recognized train of well-marked symptoms common to all these cases, in which certain structures are specially invaded, and undergo a clearly marked metamorphosis, the latter differing in its character from all other results of inflammatory action, there yet remains something to be said as to the nature and extent

of the injury which may give rise to sympathetic ophthalmia, the period at which the disease usually exhausts itself, the structures which it involves, and, finally, as to the best method of treatment to be adopted for its amelioration. The disease is, however, one which demands, and can alone be compassed by, the united vigilance and observation of all those who have had experience of its history, and we can only obtain a complete and exhaustive literature of this difficult subject in all its various aspects if each ophthalmologist who has had some special experience, founded on cases under his own immediate observation, will place his facts on record, and thus enable us gradually to acquire a complete and accurate knowledge of the disease. Influenced by these considerations, I propose, in the latter portion of this paper, to quote briefly a few cases that have come under my notice, and that seem to have a practical bearing upon sympathetic ophthalmia; and, in so doing, I follow the good example set by my esteemed friend Mr. George Lawson, who contributed a valuable paper on this subject in the last issue of the Reports, and more recently by Dr. David Webster, of New York, who related some very interesting experiences in this disease before the American Ophthalmological Society.

Regarding the nature and extent of the injury which may be the exciting cause of this disease, whilst it may be admitted that the presence of a foreign body in the eye is a frequent factor, and must be regarded as a permanent source of danger, we not infrequently find that simple wounds are sufficient to induce this terrible malady: and although wounds which invade the ciliary region are certainly the most dangerous, yet cases have come under my observation in which injuries, and even simple incisions of the cornea, have produced the same result. Mr. Brudenell Carter, in his work on Diseases of the Eye, quotes examples of this, and there are doubtless few ophthalmic surgeons of large experience who have not had cases, though fortunately such are of rare occurrence, in which an ordinary corneal

operation for cataract that gave early promise of success, has been followed by total eclipse of both eyes. I may here venture to express my individual opinion, that, whilst the nature of the injury exerts, as a rule, a very powerful influence upon the initiation of sympathetic ophthalmia, yet the fact that injuries of all descriptions frequently occur without being followed by this result, and the extreme rarity with which our operations give rise to it seem to prove that a peculiar and exceptional condition of the nervous system is a powerful agent in these cases. Further experience and investigation in this direction may, I venture to hope, afford valuable aid in diagnosing the constitutional soil in which this terrible weed is likely to grow. As to the period occupied in the development of the disease, I have usually found that it has run its active course, and completed its morbid changes, in from twelve to eighteen months; and it may thus be said to have a marked and finite limit.

When this has been reached, the affected organ may, and not infrequently does, remain unchanged for an indefinite period, showing no tendency either to improvement or deterioration. The symptoms and pathological changes which characterize this disease have been so fully described in well-known works, that I merely call attention to them in order to insist on a practical point, which, though sometimes overlooked, is of great value, namely: that, although we have in every instance a complete transformation of the iris, lens, and capsule into a tough, opaque, and inelastic tissue, yet, as a rule, the fundus of the eye is but slightly involved. As regards the general principles of treatment, it will at once be conceded that, in cases of direct injury, those in charge are relieved of all responsibility when the wound is of so severe a nature as to necessitate the immediate removal of the eye. There are cases, however, of comparatively slight injury, where excellent, if not absolutely normal vision is retained in the wounded organ, so that the question of enucleation can hardly be suggested, and would certainly not be entertained either by patient, parents, or friends; and yet

when, from the character and situation of the wound, the possible and not improbable occurrence of sympathetic ophthalmia rises like a spectre before the mind of the oculist. The correct and obvious course to pursue is to keep such cases under close observation, so that active measures may at once be adopted on the first approach of danger. If the damaged globe be removed before any organic mischief has commenced in its companion all will be well; but if disease be once sympathetically established, even in a slight degree, enucleation of the injured eye entirely fails to arrest the trouble, and more than one case has come under my observation where some useful sight has ultimately been retained in the wounded globe when that which was secondarily involved has become absolutely blind.

As to the treatment of the disease during its progress, I have never been able to satisfy my mind that we possess any means of exerting a specifically beneficial influence, either by local or constitutional remedies. We may endeavour in the very early stages to counteract the tendency to contraction of the pupil by the use of atropine, or our yet more powerful ally duboisine; but the rapid metamorphosis undergone by the iris soon renders these remedies useless and inert; whilst a too long continuation of them may do positive harm. Light should be carefully excluded, and the constitutional power of the patient maintained, but not stimulated. In these simple rules may be comprised all that our art can at present accomplish. Any operative interference during the progress of the disease is, in my opinion, contra-indicated. Every attempt that I have made, or have seen others make, in this direction has been followed by aggravation of the symptoms. It is only when the disease has run its course, and finished its work of destruction, so that the eye has not only become perfectly quiet, losing all vascularity and sensitiveness to light, but when this condition of quiescence has existed for some time, that it may become expedient to commence operative measures. In illustration of this part of my subject, and in explanation of my treatment at this

final stage, I cannot perhaps do better than quote the following cases which have recently occurred in my practice :—

The first case is that of Henry L., aged twenty-two. When ten years old he wounded his right eye with a packing needle, penetrating the ciliary region deeply and destroying the sight. Some months later, he came under my care at the Moorfields Hospital, with sympathetic inflammation of the left eye. The right eye was removed, but the disease progressed in the other, and the boy became blind. I declined to operate then, but expressed a desire to see him at a later period. He was admitted into a Blind School, and for nine years he was educated as a blind boy. At the end of that time, in December, 1877, he came to me at the Middlesex Hospital. I found the tension normal, the anterior chamber good, the iris and capsule presenting the appearance usual in these cases. There was faint perception of vivid colours but no definition of objects. The patient was admitted into the hospital, and, after he had been placed under the influence of an anæsthetic, I proceeded to perform the first operation in the following manner. Using a speculum, and fixing the globe, I introduced a fine needle through the cornea, and directed the point to the centre of the opaque capsule. This proved so tough that the point of the needle would not penetrate, and I found, that if I continued to thrust on the point, I pushed the capsule back, with a risk of dragging on the ciliary region, or separating the iris from its ciliary attachment. I therefore kept the point gently pressed on the surface of the capsule, and by making a rapid rotatory movement, on the principle of the gimblet, I succeeded in partially penetrating, and entangling the point in, the substance of the capsule. I then introduced a second needle from the opposite side, and by bringing the penetrating force of one needle to bear upon the other, I pierced the tough capsule with both : then, by separating one point from the other, I succeeded in making a rent in the centre. This was followed by the escape of a small quantity of lens matter, of the consistence of cream cheese. The

operation gave rise to very slight reaction, and an interval of two months was allowed to elapse during which the soft lens matter was absorbed, and the capsule closed again. The same operation was repeated with greater facility and with the same results; more soft lens matter escaped, but to a less amount; this again was gradually absorbed, and in the course of about two months the capsule closed once more. For a third time the same operation with two needles was performed, and, on this occasion, I was rewarded for my perseverance by obtaining a small round clear space, through which rays of light could penetrate to the retina, so that, after an interval of a few months, with a + $3\frac{1}{2}$-inch lens he saw $\frac{10}{50}$ and with a + $2\frac{1}{2}$-inch could read Jaeger No. 2. The patient has gone to America, having accepted a situation there as a church organist; but before he left in August last he was seen, and his vision tested, by Dr. Warlomont, of Brussels, who was in London at that time. Practice had improved the sight, and with the glasses for reading he could decipher a few words of Jaeger 1.

The second case is that of a child, Master Stanley D., aged eight, from Valentia, in Ireland; he met with an injury to his right eye, about five years ago, by running against the latch of a door. He was seen by Dr. Swanzy, of Dublin, who suggested enucleation, but, owing to the anxiety of the parents to save the eye, the operation was not performed. The child was taken home, and, at the expiration of three weeks, the left eye became sympathetically affected. Five weeks after the injury, the right eye was removed by Dr. Macnaughton Jones, of Cork; but the sympathetic inflammation progressed in the left eye till there was only perception of shadows. When I saw the child in April, 1880, more than four years after the injury, the left eye presented the usual appearances observable in these cases, and was in all respects similar to the one already quoted. I operated on May 19th, pursuing exactly the same plan with two needles that I have described above, and in this instance also lens matter was released from between the layers of the tough capsule.

The eye bore the operation well, but the pupil gradually reclosed, and a final needle operation on August 16th, accomplished the desired end, namely: a clear round central pupil through which the child, with a + 4-inch lens saw $\frac{10}{70}$. He had not been taught to read, but, with a + $2\frac{1}{2}$-inch lens, he distinguished the second-hand on a watch.

Some years ago I read a paper on sympathetic ophthalmia, at the Heidelberg Congress, in which I quoted somewhat similar cases, and advocated a similar method of operating; and all my subsequent experience has confirmed the soundness of the views I then put forth. On the other hand, where I have seen iridectomy, iridotomy, and extraction attempted, the result has been disappointing. As I am decidedly of opinion that the successful issue of the cases which I have cited was largely, if not entirely, due to the special method of operative procedure which I adopted, I have brought this subject forward again in the hope that my experience may prove useful to the profession.

## A SUMMARY OF CASES OF OCULAR PALSY, WITH COMMENTS.

By J. W. Hulke, F.R.S., F.R.C.S.,
*Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital.*

The interest excited by two papers on ocular palsies, recently communicated by Mr. J. Hutchinson to the Royal Medical and Chirurgical Society,* has induced me to offer to the readers of these reports a summary of 127 cases occurring in my hospital and private practice.

Of this total eighty-four are instances of palsies of extra-ocular muscles only, nineteen are examples of palsies of intra-ocular muscles alone, whilst in the remainder, twenty-four, the extra- and intra-ocular muscles were simultaneously implicated.

### Group I.—*Palsies of Extra-Ocular Muscles supplied by the Third Cranial Nerve.*

Since this nerve supplies several muscles, and many orbital palsies have a peripheral origin, it might be inferred that its palsies would be much more frequent than those of the fourth and sixth nerves distributed as these are each to one muscle only.

As regards the fourth nerve this inference is borne out by facts, but it is not so with respect to the sixth nerve, for palsy of the Rectus externus is (as, I think, was remarked by the late A. v. Gräfe) of singularly frequent occurrence.

An analysis of nineteen cases of palsy of those extra-ocular muscles, only which are innervated by the third nerve, (all other ocular muscles escaping), shows that in five of these

* Med. Chir. Trans., vol. lxi, p. 216, and vol. lxii, p. 307.

the Rectus superior alone was implicated; in three the Rectus inferior alone; in two the Rectus internus only; in one the Obliquus inferior. In the remainder eight more than one muscle, and in a few all the third nerve muscles were palsied.

As regards their origin in a very small number of cases, violence was the immediate and sole cause—as in the following instance where all the third nerve muscles on both sides except the Levatores palpebrarum were palsied.

Case 1.—A sail-maker, æt. 31 years, a spare, wiry man, in good health, fell from the ship's top-mast into the rigging, and thence on to the deck. His shipmates took him up stunned, and placed him in his berth, where he lay unconscious during several hours. On regaining consciousness he found his sight very confused, and those about him saw that his eyes were "turned."

Thirteen months after this accident, on his ship coming into port, he was brought the same day to the Royal London Ophthalmic Hospital on the 4th February, 1864. Both eyes were seen to be directed strongly outwards, the temporal border of each cornea being not more than 2mm. distant from the outer commissure of the eyelids, and by no effort could they be inverted. A strong effort to look up caused a very slight upward movement of the cornea, and the effort to look down produced an equally limited movement of the cornea downwards, jerky, and with slight rotations.

The pupils were equal; in moderately bright daylight their estimated diameter was 2·5mm. to 3mm., which under exposure to full light became about 2mm. With the left eye he read No. 8, and with the right eye No. 10 in Jäger's scale.

The left optic papilla had a pale bluish colour, the details of its lamina cribrosa were unnaturally distinct from the centre outwards nearly to the edge, and the arteria centralis was unduly small. The right optic papilla was congested.

Here the simultaneous implication of both three nerves pointed to an intracranial, and I suggest, a central cause. Assuming the correctness of this, what is the explanation of the escape of the nerve twigs to the Levat. palp. and to the intra-ocular muscles? It may be found, I think, in the experi-

ments of Hansen and Voelckers undertaken to determine the origin of the accommodation nerves.* These careful investigators discovered that in the dog the roots of the third nerve are a series of separate bundles of fibres, by stimulating each of which in turn from before backwards, movements first of accommodation, next of the iris, and then of the Rect. sup., Levator palp., Rect. int., Rect. inf. and Obliq. inf., could be provoked in the sequence here given. It is, I think, easier to conceive that the injury might have occasioned a laceration of cerebral tissue, or hæmorrhage implicating some, and sparing other roots in a situation where these are separate, than after their combination in the nerve, and after it leaves the surface of the crus.

Case 2.—A young tailoress, in withdrawing her head from an open window, struck her crown against the sash-bar. The blow was so severe that it stunned her for a few moments, and made her head ache much. Within an hour of the accident her left upper eyelid had fallen so much as to cover the pupil, which for a short time made her suppose the eye was blinded.

Three weeks after this, when I first saw her at the Royal London Ophthalmic Hospital, on 29th March, 1865, complete palsy of the Levator palp. sup., Rect. int., sup., inf., and of the Obliq. inf. was noted.

Here the palsy followed so quickly upon the injury that the causal relation of the latter was highly probable. Antecedent intracranial disease could not, however, be confidently excluded for the patient spoke of having had frequent headaches, and a neighbour hinted that she had been occasionally light-headed. There was, however, some doubt whether this was not from alcohol taken to drown care, for her circumstances were truly wretched. She and her husband were both out of work, and they were evidently insufficiently fed and clothed.

Once pregnant she had miscarried, and had ever since had profuse leucorrhœa.

Of five patients who had palsy of the Rect. sup. only, one (3rd case) was a lad of 14 years, who was brought to the Royal London Ophthalmic Hospital on 28th November 1874, at which time the diplopia was said to have existed for

* Arch. fur Ophth., Bd. 24 Abth. 1, s. 1. u.f.

about one year. It was limited still to the upper part of the visual field. When he looked up the right cornea plainly lagged below the left. No cause was discoverable, he appeared to be healthy, and had not, he said had any accident or illness. In the absence of any positive indication, nux vomica and iron were prescribed. Contrary to expectation, at the end of a month the palsy had greatly diminished.

The case just narrated is remarkable for the rapid improvement under treatment, and for the absence of contracture of the antagonist of the palsied muscle at the end of so long a time as one year.

In the following case contracture of the antagonist muscle was already present at a much earlier time.

Case 4.—A smith, æt. 38, a tall, spare, muscular fellow, after sitting in a full draught at an open window in a paroxysm of asthma, saw double. A few days after this, when he came first under my care at the Royal London Ophthalmic Hospital, he was found to have vertical diplopia with the greatest separation of the images at the uppermost limit of the field, but beginning below the level of the eyes. The left cornea lagged below the right when he looked up. He kept the left eye covered, having found that this took away the double-sight and the dizziness.

He had had, he said, a similar attack five years previously, in which he was blind for several days. As no trace of any former disorder was discoverable in the fundus oculi it was thought probable that the blindness was not due to disease of retina or optic nerve, but was merely caused by complete ptosis of the upper eyelid.

Of the three remaining cases of palsy of the Rect. sup. only, one patient was a middle-aged labourer, another a dyspeptic clerk, and the third a lady, æt. 33, who had probably acquired syphilis.

Case 5.—A well-digger who, with the exception of an ague seven or eight years previously, had enjoyed good health, came to the Royal London Ophthalmic Hospital on 12th July, 1867, for advice about a confusion of sight, which puzzled him so much that he could not work. It was plainly diplopia due to palsy of the Rect. sup. of the left eye.

He attributed his disorder, and probably correctly, to a severe cold in his forehead, with browache which came on after working during several hours in a cold well with his cap wet through with dripping water.

Case 6.—A very emotional little brunette, æt. 33, was brought to me by her family medicus in January, 1877, suffering from confused sight, dizziness, and a fixed uneasiness in the crown of her head which made her very apprehensive of brain disease. The visual trouble was diplopia in the upper half of the field due to incomplete palsy of the Rect. sup.

Her husband had had syphilis, and although whilst under the care of her present medical attendant, she had not herself evidenced any manifestation of this taint, there were circumstances in her earlier history, after marriage, which made infection not improbable.

She improved under the use of iodide of potassium.

In the following cases two muscles associated in the concerted movement of eyeball and eyelid in looking upwards were simultaneously palsied: one the Levator palp. sup. and Rect. sup.; in the other the Levator palp. sup. and Obliq. inf.

Case 7.— ——, æt. 33, a healthy-looking woman, came under my treatment at the Royal London Ophthalmic Hospital on 27th April, 1869, for double-sight in the upper half of the field at the extreme upper limit of which occurred the greatest separation of the two images.

The left cornea was manifestly lower than the right. There was also ptosis of the left upper eyelid.

Six years previously she had had, she said, an attack of double-sight obliging her to turn her head towards her left shoulder. This passed off, and sixteen months ago her left upper eyelid began to droop and in looking towards her right objects seemed doubled. At that time she had much pain in the back of the head. This attack also passed off, and she remained free from ocular trouble until shortly before her present illness.

Case 8.— ——, æt. 33, consulted me on 13th September, 1872, for diplopia which had come on suddenly a few days previously in the course of convalescence from a severe gastric disorder

which the family medicus called "*Liver*." He had had a similar confusion of sight in 1870.

Of the three patients with palsy of the Rect. inf. only, one was presumably tainted with syphilis, and another was rheumatic.

Case 9.—On 30th June, 1865, I saw in consultation a middle-aged lady for double-sight in the lower half of the visual field. She plainly had incomplete palsy of the Rect. inf. of the right eye. She had had a similar attack three years previously lasting three months, and disappearing under the use of iodide of potassium. A long illness, after marriage, was probably, from its symptoms, syphilis.

Case 10.— ——, æt. 43, dyspeptic and very rheumatic, came under my care at the Royal London Ophthalmic Hospital, 23rd February, 1863, for diplopia in the lower half of the visual field which was plainly due to paresis of the Rect. inf. of the right eye. It had lasted one month. Syphilis was denied, and a careful inquiry failed to detect any evidence of this taint.

In the two next cases palsy of the Rect. int. was presumably due to a central disorder, in the first perhaps a gumma, in the second senile softening.

Case 11.— ——, æt. 26, had a chancre in the autumn of 1865, followed by secondaries. In November, 1867, he was under treatment for iritis, and on 12th September, 1871, he again came to the Royal London Ophthalmic Hospital, now with confusion of sight which hindered him from working.

He had incomplete palsy of both Rect. int., both eyes diverged, and the vertical corneal meridian could not be brought inwards to the middle of the palpebral fissure. He had also at this time double sarcocele and condylomata around the anus.

Case 12.—A housekeeper, æt. 64, came to my polyclinic at the Middlesex Hospital on 13th January, 1872, complaining of double sight, which she had first noticed a fortnight previously. It was found to be due to incomplete palsy of the Rect. int. of the right eye. By May this had quite disappeared. In

the following December she returned, having now much pain in the left temple and deafness of both ears. She soon after died with symptoms of cerebral hæmorrhage.

In this case the ocular palsy occurred late in the course of locomotor ataxy; more often it happens at a much earlier date in this disorder.

Case 13.—On 10th April, 1868, I was consulted by a lady on account of confusion of sight which had lately intervened in the course of an illness of several years' duration. This confusion, she had herself discovered, occurred only when both eyes were open; when either eye was covered, she read her newspaper comfortably with the other, but reading with both eyes was made nearly impossible by the doubling of the type. She was found to have imperfect palsy of both Rect. int. The eyes were divergent and the inward range of the eyeball was very restricted.

She had been confined to her couch for many years by a disorder which began with pricking and numbness of the soles of the feet which made her gait unsteady, and gave rise to the unfounded suspicion that she drank secretly. Some time later she could stand and walk only whilst her eyes were fixed upon her feet. The numbness gradually crept upwards from the soles, and at the time when I saw her the whole surface of her body up to the level of her collar-bones was insensible to tactile stimuli. The muscular power of her limbs was not appreciably weakened, and she could even perform concerted actions, such as taking up her spectacles off a table and putting them on without help; she could also hold her newspaper, but whilst doing this she was obliged to keep her eyes fixed upon the object, for when she took them off, the spectacles or paper fell from her hands. Muscular sense was very defective, she had no correct idea of the force she used, and had, I was told, on more than one occasion crushed the stem of a wine glass which she thought she was holding lightly.

Although it was several years since she had tried to walk, her legs still retained considerable muscular power. She was seldom free from sharp darting pains.

Exposure to cold, which appeared in two of the above cases to be the exciting cause, was probably operative in the

following case where all the third-nerve muscles were involved.

Case 14.—A plasterer, æt. 40, after working in a draught, felt violent pains in the left temple, and the same night for half an hour he had "fearful" pain behind the left eyeball. Next morning he found that the upper eyelid had fallen, and he saw double. Eleven days after this, on 13th May, 1862, when he came under my care at the Royal London Ophthalmic Hospital, of all the muscles of the left orbit, only the Rect. ext. and Obliq. sup. were unpalsied, all the others were absolutely palsied.

The man was feeble, he had had hæmoptysis, and was very liable to headache.

Under the use of iodide of potassium and faradisation the palsies completely disappeared.

Group II.—*Palsies of Muscles supplied by the Third Cranial Nerve concurrent with Palsy of other extra- and intra-ocular muscles.*

In the two following cases palsy of extra-ocular muscles supplied by the third nerve was associated with anæsthesia in the distribution of a transorbital twig of the fifth nerve. The sole cause discoverable in one was an injury, whilst in the other case syphilis was probable.

Case 15.— ——, æt. 38, healthy mother of four healthy children, stumbling in the dark struck her brow against a piece of furniture, slightly cutting the free border of the upper eyelid. A fortnight afterwards, when I first saw her, a small scar marked the injury in the lid, and in the conjunctiva over the insertion of the Rect. sup. She had absolute ptosis of the upper eyelid and complete anæsthesia in the distribution of the supra-trochlear branch of the first division of the fifth nerve. The mobility of the eyeball was perfect. When last seen, five months later, the ptosis was less, and the anæsthesia nearly gone.

Case 16.—A brickmaker, æt. 42 years, came to my polyclinic at the Royal London Ophthalmic Hospital on the 18th

November, 1876, complaining of dizziness and troubled sight, which had come on about six weeks before with pains in the right side of his head, in the neck, and between his shoulders. These he had thought rheumatic, as his work exposed him much in all weathers. They were followed shortly by drooping of the upper eyelid, which in eight or nine days had completely fallen.

He had palsy of all the third-nerve extra- and intra-ocular muscles and anæsthesia in all the transorbital branches of the first division of the fifth nerve.

He had had a chancre twenty-four years before, and of thirteen children his wife had borne three only survived.

In the case now to be related all the extra- and intra-ocular muscles were implicated. Here, too, syphilis was probable.

Case 17.—On May 11th, 1872, a thin, pale-faced, trembling woman, æt. 46 years, came to my polyclinic at the Royal London Ophthalmic Hospital for advice about her left eye. Its optic nerve was in a condition of advanced atrophy. All the muscles (third, fourth, and sixth nerves) were palsied, the pupil, semi-dilated, did not act consensually with that of the right eye in response to light, nor in concert with the accommodation of this eye. The surface of the eyeball and of those parts of the face and scalp innervated by transorbital branches of the fifth nerve were insensible to tactile stimuli. The left ear was deaf, but it was uncertain when hearing first failed.

The patient related that her illness had begun about seven months previously with great pain in the back of her head, and that this was soon followed by drooping of the upper eyelid. Her husband was a sailor often long absent from her. She had borne six children which only survived a few days.

In spite of the free exhibition of iodide of potassium, so useful in similar cases, the affection of the fifth extended to the second and third divisions, and in the following September when last seen ophthalmia and ulceration of the cornea had supervened.

In the preceding case "Ophthalmoplegia Externa," a term which is applied to palsy of all the extra-ocular

muscles was associated with anæsthesia of the fifth nerve. The condition was limited to one side, and the woman's history since marriage made syphilis extremely probable. In the next case every extra-ocular muscle in both orbits was palsied, the intra-ocular muscles escaping; here no evidence of syphilis was present. The patient's manner was frank; he was under my observation during several months; he denied ever having exposed himself to the possibility of infection. No scar of primary sore was discernible, and no history of throat or skin affection could be elicited. His wife, whom I also saw several times, was, and had always been, healthy. She had never had any symptoms which could lead to suspicion of syphilis, and their children (four) were remarkably healthy. Under these circumstances, I submit, the notion of a syphilitic origin of the palsies is untenable, notwithstanding his recovery under iodide of potassium.

Case 18.—On December 15th, 1869, I admitted as an in-patient into the Middlesex Hospital, a tall, muscular, hale-looking carpenter, in order that I might be able to watch him more closely. He had been already at that time for fourteen weeks an out-patient with progressive palsy of the orbital muscles concurrent with a difficulty in walking, which he attributed to numbness of his legs.

When admitted he had absolute palsy of all the extra-ocular muscles of the right side, and nearly complete palsy of all those of the left orbit. The right eyeball was absolutely immovable, and the left eyeball nearly so. Sight was unaffected, each pupil was active, and accommodation corresponding to his age. The fundus oculi had a perfectly normal appearance. He had much headache. His gait was slow and deliberate, with an unsteady balancing manner suggestive of locomotor ataxy. He could, however, stand and walk with his eyes shut. A week later he said that his arms and legs were numb; and this was not merely a subjective sensation, for trial with compass points bore out his statement.

Under full doses of iodide of potassium perseveringly taken during several months the symptoms slowly and completely disappeared, and he was ultimately able to return to work as a

carpenter. When I last saw him, he walked naturally—the distance from Islington to the Hospital, nearly three miles, had not fatigued him—and the mobility of his eyes was perfect.

The number of patients in whom the ciliary muscle and iris were palsied concurrently with one or more of the third nerve extra-ocular muscles was about one-fifth greater than that of the extra-ocular third nerve palsies alone. Syphilis in the intra-ocular palsies was also a fertile cause.

Case 19.— ——, a clerk, æt. 31, steady of late, since marriage, but previously very "*gay*," came under my care at the Royal London Ophthalmic Hospital on 26th April, 1879, for great confusion of sight, which had begun seven weeks previously, and now obliged him to leave off work. He had complete ptosis of the upper lid, and a strong outward squint of the right eyeball, and the extra-ocular third nerve muscles were absolutely palsied. The pupil was widely dilated and motionless, and accommodation was lost. He was drowsy, odd-mannered; he had slight transient hallucinations, and much headache. On his leg was a characteristic tertiary ulcer.

Slight mitigation of the headache, and slight restoration of mobility of the eye followed the use of iodide of potassium, but the improvement was only transient; he soon relapsed and he died in the country in the course of the summer.

The next is very similar to the last case; the evidence of syphilis is scarcely less clear, the interval between the date of infection and the ocular symptoms probably not widely different. The suddenness of the attack and rapid yielding to iodide and recurrence of palsy are not without interest.

Case 20.— —— a gamekeeper, æt. 40, came under my care at the Middlesex Hospital on 10th May, 1867. He had then absolute ptosis of the upper eyelid and palsy of all the third nerve extra-ocular muscles of one eye; the pupil, too, was dilated and motionless, and accommodation was lost.

He said that, eight weeks before, whilst shooting, he became giddy, everything around him looked confused, and soon things seemed to him to be doubled. For four years previously he had

had severe browache, much sleeplessness, frequent vertigo, and nausea. Eight years before the present ocular trouble, he had had a chancre, followed by ulcerated throat.

Under the use of iodide of potassium he made a quick recovery, but he returned in the following November with complete palsy of all the muscles implicated in the previous May; he had now also much pain in the temple and occiput, and nausea. He was again ordered iodide. His further history is unknown to me.

In the following case the evidence of syphilis is scarcely less strong; an acknowledgment of primary sore, without, however, evidence of secondaries.

Case 21.— ——, æt. 30, was suddenly seized with giddiness and confusion of sight, and soon after this his upper eyelid was seen to have fallen.

Eleven days after this he sought advice at the Royal London Ophthalmic Hospital. He had then slight ptosis of upper lid, and impaired action of all the third nerve muscles of one eye. Its pupil also was dilated and motionless, and there was loss of accommodation.

His appearance was healthy, and he affirmed that he had only once been ill, and that a slight temporary indisposition, during the preceding eleven years. On pressing him, however, he acknowledged a chancre six years before the present attack.

In the next case, the interval between the primary sore and the ocular palsy—19 years—was so great that a causal relation might be doubted, but the great mortality of the children strengthens the suspicion.

Case 22.— ——, æt. 42, a feeble, sallow-faced man came under my care at the Royal London Ophthalmic Hospital, on 29th April, 1879.

He saw, he said, two things where he knew only one to be present; he was dizzy, and he often retched.

He was found to have imperfect palsy of the Rect. sup., and mydriasis, and a few days later the Rect. int. was implicated.

During the two immediately preceding years he had had, he said, several similar attacks, which had quickly yielded to treat-

ment. In 1872 he was jaundiced, and since then he had often bilious vomiting. Nineteen years ago he had had a chancre. Soon after which he married. His wife had borne to him twelve children, of which the seven first all died.

The ocular palsies disappeared under the use of iodide of potassium.

## ON THE CONTINUOUS ELECTRICAL CURRENT AS A THERAPEUTIC AGENT IN ATROPHY OF THE OPTIC NERVE AND IN RETINITIS PIGMENTOSA.

By R. Marcus Gunn, M.A., M.B.,

*Clinical Assistant, Royal London Ophthalmic Hospital.*

### I.—*In Optic Atrophy.*

All the cases in which the continuous current was used are here brought forward, with the exception of three: two of these were treated for too short a time to be of any value, and the remaining one was a case of optic neuritis, in which the current was employed during the stage of swelling only.

I must apologise for the frequent imperfections of my notes. In the hurry of daily routine-work observed facts were often left unnoted, and important questions unasked.

*Method of application.*—I used Weiss' continuous current battery* of twenty-five cells; it was fitted in the ordinary way with sponges, which were moistened in a weak solution of common salt. On employing it for the first time in any case the positive pole (anode) was applied lightly over the closed lids of one eye, and the negative (cathode) to the opposite eye or temple. The power was now gradually increased, beginning with five or seven cells, until a distinct sensation of a flash of light was experienced by the patient on making or breaking the circuit.† The anode was now kept over the eyelids, while the cathode was applied in turn to the top of the spine, to a point just behind the mastoid process, and to the supra-orbital region of the same side, so as to determine which position gave the greatest light-impression. This varied in different individuals, but in the majority of cases the supra-orbital region was selected. This position (*e.g.*, the supra-

* The Foveaux-Smee. The elements are zinc and platinised silver.

† On the characters of the light sensation produced on the healthy optic nerve, see Helmholtz. (Physiologische Optik. Leipzig, 1867, p. 204.)

orbital) being determined, the cathode was kept there for half-a-minute or so, then removed for a few seconds, and finally reapplied for the same period as before. It was next moved to the corresponding point on the opposite side of the head, and then to the closed eyelids of this side; in both these positions it was applied for a few seconds. Both poles were now removed, and the cathode then applied where the anode had been, while the anode was applied to the supra-orbital region, etc., as above.

The same course was then gone through with the other eye, a slightly stronger current being finally used for a few seconds. The whole sitting lasted from five to eight minutes.

The point of selection was determined once for all at the first sitting, and noted.

CASE I.—*Symmetrical failure of ten months' duration. Cause obscure. Improvement immediate, but temporary.*

Charles K——, a Swiss, æt. 37, married. Admitted February 6, 1877, under the care of Mr. Critchett. A pale, thin man of medium height, hair dark brown, irides grey; intelligence very good.

*History.*—Habits temperate as to alcohol and tobacco. He has been subject to rheumatism for many years. Doubtful history of syphilis. His sight began to fail about ten months ago. He was then, and is still, a sufferer from severe headaches.

No abnormal spinal symptoms. Tendon-reflex not tried. $V = \frac{2}{70}$ with each eye. Colour-perception defective: he calls *green*, "clear red;" *orange*, "yellow;" *black*, "grey;" *crimson*, "clear red." Blue, violet, yellow, and white he names correctly.*

*Ophth. Exam.*—Both optic discs are white and atrophied.

Ordered nux vomica and iodide of potassium. To have the continuous current daily.

February 12. $V = \frac{8}{70}$.

,, 20. $V = \frac{9}{70}$.

* Holmgren's wools were not in use at this time at Moorfields.

February 25. V = $\frac{10}{70}$ and "P" of $\frac{10}{50}$.

" 28. V = $\frac{11}{70}$.

On March 15th he was transferred to another hospital. There his sight again deteriorated, and he returned in about two months as an out-patient here, with his vision much as at first. He had been having the battery, but he says "it was not used in the same way."

He attended at intervals for some time as an out-patient, and had strychnine and phosphorus pills, but his sight gradually grew worse. At his last visit he could not count fingers.

While in the hospital, when tested a few minutes after the use of the battery, his vision was always better than before its use; but this marked improvement did not last above an hour or so. The real daily improvement was very small although quite positive.

CASE II.—*Symmetrical failure of three months' duration. Rather intemperate in tobacco and beer. ? Syphilis. No decided improvement.*

Charles G——, æt. 25, unmarried. Admitted October 16, 1877, under the care of Mr. Soelberg Wells. Family history unimportant. Patient has always been a strong healthy man. For the last seven years he has smoked a daily allowance of about half-an-ounce of tobacco (cut cavendish). He drinks three or four pints of beer daily. Seven years ago he had what he calls a "soft chancre:" no history of sore throat or secondary eruption. About three months ago his sight began to fail in both eyes. At first there was merely dimness, but this gradually grew worse for about a month, when his condition was as at present.

No marked abnormal spinal symptons.

Pupils sluggish. Irides irregularly pigmented. Tension normal.

*Right eye.*—V = Bare perception of strong artificial light and limited to lower part of field.

*Left eye.*—V = Perception of medium light—best in lower and inner part of field. Cannot see objects.

*Ophth. Exam.—Right.* Disc greyish in colour with absence of capillaries. Arteries reduced in size. Peculiar distribution

with much intertwining of large vessels. Deep physiological cupping.

*Left.* Disc pale. Arteries small. Deep physiological cup. Below the disc there is considerable retinal degeneration (apparently fatty with cholesterine) and hypertrophy of arterial sheath.

*Treatment.*—Subcutaneous injection of strychnine daily, quantity gradually to be increased.

Iodide of potassium in mixture. To have the continuous current daily.

November 14.—*Right.* V = Bare perception of hand-reflex as "something white."

*Left.*—Just sees hand in axis of vision, but nowhere else.

November 16.—The physiological action of strychnine was now very marked, and the injection was discontinued.

November 27.—Tonsils enlarged and superficially ulcerated.

December 8.—V as on last examination. No certain improvement.

December 19.—Discharged.

The very slight improvement which occurred in this case was possibly due to the action of the strychnine.

Case III.—*Gradual symmetrical failure of three years' duration, following injury to head. No improvement.*

Michael P——, æt. 32, widower. Admitted November 14, 1877, under the care of Mr. Wordsworth. A well-nourished man with rather florid complexion. Adherent ear lobes. Intelligence good.

*History.*—He has always been healthy and temperate. Has worked very hard as fireman at a stationary pit-engine, generally twelve hours a day. In January, 1872 (nearly six years ago), he had a severe injury while employed attending a travelling crane; both legs were broken, and there was a deep wound over the right eyebrow. He lost consciousness and remained unconscious an hour, and soon relapsed into this condition for seven hours. He "wandered a little" in his sleep during that night, and had severe pain in the forehead for several weeks. About three years ago he first noticed dimness of sight in both eyes equally, and at this time he was always very drowsy. The sight gradually failed until September, 1875, when he had to give up work. Since November, 1875 (two years ago), his sight has

been as now. He has never had any pain during the failure of sight except an occasional "shoot" through the head. Last summer he first noticed diplopia. His other special senses have been, and are, perfect. No history of syphilis.

*Pres. condition.*—There is a deep scar over the right eyebrow, and a patch of grey hair at the part of the head directly opposite. (The latter was noticed about two years after the accident.) Convergent strabismus of left eye. Paresis of both external recti, better marked in left. Pupils semi-dilated and quite inactive. Nystagmus: quick horizontal jerkings. No pain in eyes. Tension normal.

V = Perception of large objects with each eye.

*Right.*—Field limited peripherally and equally all round.

*Left.*—Peripheral limitation all round, but most marked outwards and downwards.

*Ophth. Exam.*—Media clear (examination difficult on account of nystagmus). Both discs are bluish white with atrophic cupping (or rather *saucering*, if I may be allowed the expression). Vessels about normal size: arteries rather small. At or near right macula is a whitish patch. Fundus otherwise looks normal.

*Treatment.*—Nux vomica in mixture. Continuous current daily.

He describes a large blue flash on making, and a small "darker flash" on breaking the circuit. He required twenty-one cells.

December 8.—V = Bare perception of hand moving at about 8 inches with each—better with right.

December 17.—Discharged.

Case IV.—*Great failure of vision of fourteen months' duration. History of injury to head and of syphilis. ? Locomotor ataxy. No improvement.*

Frank D——, æt. 38, married. Admitted November 27, 1877, under the care of Mr. Lawson.

Born in England, but has been for several years a farmer in Arkansas. He is one of a family of fourteen. A younger sister died suffering from exophthalmic goitre.

About thirteen years ago he had a severe fall from horseback in the hunting-field, pitching on his head. He was not insensible, but he had great pain in the neck for some time after this

injury. A year later he contracted syphilis (secondary eruption, sore throat). About sixteen months ago, while exposed sleeping out at night, he had a severe attack of quinsy, which lasted for about six weeks before the abscess burst. Directly afterwards he felt a numbness in the jaws, and closing the teeth gave him pain. This numbness, he says, gradually crept up his cheeks, affected his temples, and then his sight began to fail,—the right about a month before the left. His wife said that the left eye was at first turned in and he saw double images in the distance. The numbness still ascended until it reached the vertex, where it remained for some time. It is now chiefly confined to two small areas which can be covered by the finger-tips, situated about four inches from one another, one on either side of the median line of the skull and corresponding to the parieto-occipital suture. Pressure on these points causes pain.

The soles of his feet next got numb (about seven months ago), with shooting pains from the heels upwards to the knees. His feet are frequently very cold. About three months ago, he one day had a peculiar sensation in his left arm, "as if it were paralyzed," and he could not carry any heavy weight on it. He has pain on pressure over the left clavicle, corresponding to position of brachial cords. His sight has failed very gradually, with pain along the top of the eyeballs, in the temples, and across the forehead. For the last six months his legs have felt weak, and with his heels together he is very shaky, more so on shutting his eyes. He has no pain in the back, nor feeling of tightness round the waist. Tendon-reflex not tried.

He is a healthy looking man, above middle height, married for last ten years, and has one child eight years old. His health has been good, and his habits are temperate. He used to smoke in moderation.

His eyes look normal: emmetropic. Tension normal.

V = Bare perception of shadows and of hand-reflex at a few inches; better with left than right.

*Ophth. Exam.—Right.* Disc very white. Arteries diminished in size. Veins have thickened sheaths. Lamina cribrosa well marked; macula normal.

*Left.* Disc very white. Arteries small. Both arteries and veins have thickened sheaths. Lamina cribrosa as in right.

*Treatment.*—Iodide of potassium and nux vomica in mixture. Continuous current daily.

December 8.—V certainly not improved. He has less trembling, and walks more firmly.

December 31.—Condition unaltered. Discharged.

Case V.—*Much failure of vision in left eye (less in right) of six months' duration. Moderate smoker. ? Locomotor ataxy. Marked improvement.*

John G——, æt. 23, married. Admitted January 8, 1878, under the care of Mr. Lawson.

A fair-haired, short, well-nourished man. Has been married for a year. No family. No history of syphilis. He used to smoke an ounce and a half of shag tobacco weekly, but left it off on advice a month ago. It made him spit a great deal, and caused him to be very "shaky," especially lately.

Six months ago he first noticed failing sight in the left eye; but in the right only fourteen days ago.

He has weakness in the legs, with aching and pricking. Stands with difficulty with eyes closed. On each tragus there is a small, hard nodule: history of gout rather doubtful.

Pupils are now dilated under atropine. They are said to have acted "irregularly, but not to light," previously: probably with accommodation. Eyes emmetropic.

Tension normal.

*Right.*—With + 10″ V = small words of Jaeger 6 at about 6″.

*Left.*—V = Bare perception of hand. Not improved by a lens.

*Ophth. Exam.—Right.* Disc very pale, with much thickening of sheaths of vessels, especially of arteries. At inner periphery of fundus are numerous small light-coloured spots of degeneration, ? retinal.

*Left.*—Disc greyish. Sheaths very much thickened, in some quite up to the periphery. One yellowish-white spot between disc and macula. By daylight both optic discs look greenish.

*Treatment.*—Nux vomica in mixture. Continuous current daily.

January 21.—*Right.* Reads small words of Jaeg. 1 with difficulty.

*Left.*—Counts fingers at 5″. (Best without any glass). Discharged to-day.

CASE VI.—*Symmetrical failure ten weeks ago. Syphilis. Marked improvement.*

Alexander W——, æt. 29, married. Admitted May 24, 1878, under the care of Mr. Wordsworth.

A short, fairly well-nourished man, with brown hair and greyish blue irides. Intelligence very good.

About eighteen years ago, *i.e.*, when about eleven years old, he had what he believes was a chancre. (From the history he gives this is more than probable.)

Last September (eight months ago) he was laid up with severe pain all over his head, occasionally shooting into his eyes, and preventing him from getting sleep. After being off work for about two months he returned to it (as carpenter) on board ship. Shortly afterwards his headaches recommenced and lasted until about a week ago. Until about eleven weeks ago, he was subject to frequent attacks of syncope. His sight began to fail about ten weeks ago: he did not notice one eye before the other.

*Pres. Condition.*—Palpitation. Muscular tremblings. No ataxic symptoms. Occasional twitching in arms during sleep. Has sometimes had a feeling like a "tight drawing," round his right side.

Pupils barely act to light. Tension normal.

*Right.*—V = Barest perception of the gas-flash (*i.e.*, the gas turned suddenly full on and then turned off again).

*Left.*—V = Fingers at one foot.

*Ophth. Exam.—Right.* Disc greyish white. Vessels small: well marked sheath outline. Margin of disc rather indistinct. Retina looks hazy, with a few yellowish-white dots near the macula. H $\frac{1}{16}$.

*Left.*— Disc pale, margin indistinct. Vessels rather small, with sheath outline in both arteries and veins. Retina somewhat opaque. H $\frac{1}{12}$.

*Treatment.*—Iodide of potassium and nux vomica in mixture. Continuous current daily.

June 2.—*Left.* Counts fingers at 6 feet against a dark background. Reads letters of Jaeger 18 at 7″.

June 5. *Left.* Counts fingers at 10 feet. Letters of Jaeg. 16.

,, 13. Doses of nux vomica and iodide of potassium increased, now takes 13 minims of former and 10 grains of latter thrice daily.

,, 14. *Left.* V = Words of Jaeger 16 and $\frac{13}{200}$.

*Right.* V = Bare perception of hand.

,, 25. To have 16 minims of nux vomica and 15 grains of iodide three times a day.

July 8. *Left.* V = $\frac{17\frac{1}{2}}{200}$. Jaeg. 16 and very nearly Jaeg. 14.

Oct. 2. *Left.* Reads Jaeg. 14.

*Right.* Sees hand-reflex.

,, 18. V as on last trial. Discharged.

CASE VII.—*Failure of sight in both eyes (old in left). Myopia. Papillitis. Improvement.*

Michael R——, æt. 29. Admitted May 25, 1878, under the care of Mr. Streatfeild.

Family history unimportant. No history of gout, rheumatism, or syphilis. Used to have pain in testicles.

Three years ago he first noticed that the sight of the left had failed. He went to another hospital where condition was then diagnosed as neuritis. For the last three months or so, the sight in this eye has improved, while that in the right has been growing worse.

General health fairly good. No headaches.

Pupils active. Irides greyish blue, with dark hazel spots. Tension normal.

*Right.*—With − 9″ V = $\frac{20}{100}$ and 'L' of $\frac{20}{70}$. Reads Jaeg. 4 at 6″.

*Left.*—With − 9″, and V = $\frac{20}{200}$. Reads Jaeg. 6 at 4″. Letters look jumbled and distorted.

*Ophth. Exam.—Right.* Disc much injected and rather hazy. To outer side is a linear puckering of retina.

*Left.* Disc hazy, ill-defined. Veins large and dark. There is a white horizontal line of retinal puckering (linear detachment) just skirting lower edge of disc and running more or less directly

outwards and inwards to the periphery, where it becomes lost. Near the peripheral parts of the fundus, especially out and in, are extensive changes in choroid and retina. Floating bodies in vitreous.

*Treatment.*—Iodide of potassium. Continuous current daily.

June 2. *Right.* Reads Jaeg. 4 at 5″.
,, *Left.* ,, Jaeg. 1.
,, 8. Ordered nux vomica. To have continuous current every second day now, as he complains of headache following its use.
,, 12. Near vision as at last examination. Distant vision has not improved since admission. Discharged June 13.

CASE VIII.—*Great loss of vision in both eyes of long duration. Rheumatism. No improvement.*

Eliza F——, æt. 23. Admitted July 24, 1878, under the care of Mr. Streatfeild.

Mother suffers much from rheumatism or rheumatic gout. Patient has rheumatism in "every joint."

Her sight began to fail about four and a half years ago: the left eye diverged soon afterwards and has remained so. Has had occasional pain in temples and occiput since failure of sight began.

*Left eye.* Divergent, pupil sluggish. V = Bare perception of hand.

*Right.* V = Fingers at 5″. Irides greenish grey with hazel dots.

*Ophth. Exam.—Left.* Disc very white, with much diminution of vessels.

*Right.* Disc atrophic, with yellowish-white degeneration at macula.

*Treatment.*—Nux vomica and iodide of potassium internally. Continuous current daily.

August 22. V as before. Ordered tincture of nux vomica and tincture of perchloride of iron, of each, 15 minims three times a day.
,, 28. V as on admission. Discharged.

CASE IX.—*Symmetrical failure nine months ago. ? Tobacco and Alcohol. Rheumatism. No improvement.*

John R——, corn-porter, æt. 44, married. Admitted July 29, 1878, under the care of Mr. Streatfeild. Family history unimportant. Patient is a healthy looking man with ruddy complexion, light flaxen hair, and light coloured irides. He has often to lift very heavy loads at his work.

Health has always been good, with the exception of rheumatic fever twelve years ago. No history of syphilis. He drinks four or five pints of beer daily, and smokes two ounces tobacco (twist) weekly.

About nine months ago his sight began to fail in both eyes simultaneously. At first they watered much, but he had no pain in them nor in the temples. No symptoms of spinal affection.

Pupils active. V = Jaeg. 20 with each. Colour perception good.

*Ophth. Exam.*—Discs decidedly pale. Vessels slightly diminished in size, especially in right.

August—2. Ordered solution of perchloride of mercury and iodide of potassium.

,, 7.—Continuous current daily, and nux vomica.

,, 21.—V as on admission. Discharged.

CASE X.—*Failure of vision during last year. Syphilis. Temporary improvement.*

William F——, bricklayer, æt. 31, married. Admitted September 30, 1878, under the care of Mr. Lawson. He was in the army from 1868–75, and while there contracted syphilis.

He is rather pale and nervous. Light hair and complexion. Complains of sleeplessness; and has had severe headaches occasionally lately.

Sight began to fail about a year ago. It grew gradually worse, and last April he was unfit for work.

Pupils dilated and inactive. Tension normal. Emmetropia.

*Right.* V = $\frac{20}{200}$.

*Left.* V = Bare perception of hand reflex.

*Ophth. Exam.—Right.* Disc greyish white. Vessels about normal size, perhaps slightly diminished. Retina opaque looking.

*Left.* Disc as in right, but retina clearer. Thickened vascular sheath well marked near disc.

*Treatment.*—Bromide of potassium and tincture of hyoscyamus at bedtime. Iodide of potassium in mixture. Continuous current daily.

October 8. Much pain in forehead.

,, 14. Pain has extended to temples. Relieved by Heurteloup's artificial leech.

October 16. Ordered nux vomica.

,, 17. Had severe pains in head again yesterday; relieved by Heurteloup.

October 28. *Right.* V = $\frac{20}{70}$ perfectly.

*Left.* V as before.

November 1. *Right.* V = $\frac{20}{70}$ and 'P' of $\frac{20}{50}$.

*Left.* V as before. Discharged.

Re-admitted, February 11, 1879.

*Right.* V = $\frac{20}{200}$. *Left* V. = Barest perception of light.

So both have deteriorated. He is quite unable to distinguish colours, only describes their depth. He is in a highly nervous condition, and feels his position acutely, often shedding tears.

Ordered nux vomica, iodide of potassium, and continuous current as before.

April 10. He has occasionally complained of headache and giddiness. No evidence of spinal disease. At first he thought his sight was improving again, but lately it has become worse.

*Right.* V not $\frac{20}{200}$ quite. Discharged.

Case XI.—*Failing sight of three years' duration. ? Locomotor ataxy. Considerable improvement.*

Edward H——, æt. 44. Admitted October 1, 1878, under the care of Mr Lawson. Health failed five years ago while on the Gold Coast. No history of syphilis. Has smoked two ounces

weekly. Subject to flying pains all over his body. No other ataxic symptom noted. Has not had headache, with the exception of occasional slight pain in temples.

The failure of sight commenced three years ago, while suffering from a severe attack of jaundice.

For last six months sight has been as now.

Pupils inactive.—Tension normal.

*Right.* V = $\frac{10}{200}$. *Left.* V = Barest perception of light.

*Ophth. Exam.*—Discs very white with atrophic cupping. Arteries small. Veins normal or slightly diminished.

*Treatment.*—Potass. iodid. and nux vomica.

Continuous current daily.

October 28. *Right.* V = $\frac{19}{200}$. *Left.* V as before.

December 21. Battery discontinued for a few days just now. Has had considerable pain round left eye for last few days. Thinks it was caused by prolonged opthalmoscopic examination.

Dose of iodide and nux vomica increased.

December 27. Has still neuralgia on left side.

„ 31. *Right.* V = $\frac{20}{70}$. *Left.* V = Bare perception of hand reflex.

February 13. *Right.* V = $\frac{20}{70}$. Hm. $\frac{1}{20}$. *Left.* V = hand-reflex.

He confuses red, green, and yellow: calls them all yellow of different shades. Recognises blue and pink. Discharged February 15, 1879.

Case XII.—*Gradual failure for last nine months. Locomotor ataxy. Doubtful Improvement.*

Charles K——, æt. 41. Admitted December 9, 1878, under the care of Mr. Lawson. No history of syphilis. Sight has failed gradually, without pain in the head, for the last nine months or so; and he had to give up work in consequence a fortnight ago. He has shooting pains in the front of the leg, from the knee to the ankle. He staggers somewhat on closing his eyes when standing with heels together. No tendon-reflex. No feeling of distinct abdominal constriction.

*Right eye.* Pupil small and inactive. V = Barest perception of light. Tension normal.

*Left eye.*—Pupil larger than right; acts momentarily to light, then dilates and oscillates. V = Fingers at 5 feet. Tension normal.

*Ophth. Exam.*—Both optic nerves are atrophic, especially the right. Larger vessels normal. Slight "saucering" of discs.

*Treatment.*—Nux vomica. Continuous current daily.

December 31. *Right.* V as before.

*Left.* V = Fingers at 8 feet.

January 9, 1879. Discharged. V as on last examination.

CASE XIII.—*Gradual recent failure of sight. ? Locomotor ataxy. Slight improvement.*

Alexander S——, æt. 32, unmarried. Admitted December 19, 1878, under the care of Mr. Streatfeild. About seven years ago he had a fall from a top-gallant yard, and was unconscious for some days. He soon recovered from this accident, but two years later he fell from a royal yard, and fractured his thigh. On this occasion he was unconscious for eight days. No history of syphilis.

Last February (ten months ago) he first noticed dimness of sight and sparks before the eyes. For the next six months his vision failed gradually and painlessly; for the last four months it has been as now.

On being tested with eyes closed, he rocks considerably. No tendon-reflex.

V = Bare perception of light, rather better with left.

*Ophth. Exam.* Discs white and "saucered." Large vessels normal.

*Treatment.*—Nux vomica and iodide of potassium in mixture. Continuous current daily.

February 10, 1879. V = Hand-reflex, rather better with left. Discharged.

CASE XIV.—*Rapid failure of sight in both eyes. Nature obscure. ? Early Locomotor ataxy. Arrest of failure in one eye, but no improvement.*

John J——, æt. 25, single, farm-labourer. Admitted March 12, 1879, under the care of Mr. Streatfeild. Father died young:

suffered from abscesses. Of the family, two are dead; one brother alive, who is quite strong.

Patient does not smoke, and only drinks moderately. No history of syphilis, nor of injury. Has "rheumatism" in knees. Sight has been failing for five months in the left, and for two months in the right eye. He has had slight temporal pain occasionally. Tendon-reflex very good; ? exaggerated. Stands well with eyes closed. A thin, healthy-looking man: teeth good. Hair dark auburn: light hazel irides.

*Right eye.*—Pupil active. Tension normal. V = $\frac{20}{200}$ and Jaeg. 8 at 6." Optic disc pale, with deep physiological cupping.

*Left eye.*—Pupil rather sluggish. Tension normal. V = Bare perception of light. Disc pale, with deep physiological cupping, venous pulsation. Film in vitreous over vessels to inner side of disc.

He fails to recognise the colours *blue* and *red* (the latter he calls green) with right eye. The left has no colour-perception.

*Treatment.*—Ordered nux vomica and iodide of potassium.

Continuous current daily.

March 28. Has severe frontal headache and weight in eyelids. Dose of iodide of potassium increased.

April 7. *Right.* V = $\frac{20}{200}$ and Jaeg. 6 at 6" barely.

*Left.* No perception of light.

Has relaxed sore-throat. Ordered tincture of perchloride of iron.

May 3. Discharged. V as on last examination.

CASE XV.—*Old post-papillitic atrophy. History of injury to head. No improvement.*

James F——, æt. 14. Admitted April 23, 1879, under the care of Mr. Lawson. About two and a half years ago, while exercising on the bar (at the Naval School, Greenwich), he fell, alighting on his vertex. The height was about 4 feet and underneath was a hard mattress. He was unconscious for two hours, and for the next week he had pain all over his head. He says a "thick mist" used to come repeatedly before his eyes, and each time it left the "fog" thicker. This failure of sight was first noticed three months after the injury.

Six months after the above accident (March, 1877), he was admitted as an in-patient at Moorfields, under the care of Mr. Bowman, in the following condition :—

*Right eye.* Spasm of lower orbicularis fibres. Tension normal. Field good.

V = Jaeg. 6 at 6″.

*Left eye.* Tension normal. Field good. V = Jaeg. 20 at 6″ barely. Colour perception good in each.

*Ophth. Exam.*—Both discs are swollen; the veins large, dark, and tortuous.

*Treatment.*—Iodide of potassium two grains and a half three times a day.

His sight slightly deteriorated, and severe headaches, with morning sickness and vomiting appeared.

On April 21, 1877, he was transferred to the care of Dr. Hughlings Jackson, at the London Hospital. At that date my note is :—

*Right.* V = Jaeg. 8 at 4.″ Disc much swollen; at its lower part is a peculiar apparent obstruction in a vein.

*Left.* V = Jaeg. 16 at 4″.

On re-admission now, two years later, his vision is somewhat worse than on last discharge. He is in perfect health, and well-nourished.

*Right eye* slightly divergent now : has been so for more than a year.

*Right* (under atropine) with + 10″ V = Jaeger 20 letters.

*Left* „ „ with + 10″ V = Jaeger 19 *barely.*

*Ophth Exam.*—Disc white, with irregular margins. Vessels diminished in size, especially in left. There is perivascular thickening in left.

*Treatment.*—Nux vomica and potass. iodid. internally. Continuous current daily.

June 5. V not improved. Discharged.

Case XVI.—*Recent failure of sight. Probably Renal Disease. No certain improvement.*

George B——-, labourer, æt. 25, admitted June 16, 1879. About fifteen months ago he had a "chancre"—probably soft. His sight began to fail eight months ago, while he was suffering from renal dropsy—"the result of a chill."

He now suffers much from headaches, across the forehead and behind the ears. Health good (no note of urine). Hair dark brown: irides greyish-blue.

*Right eye.* Pupil fairly active—rather wide. Tension normal. V = Fingers at 2 feet, at inner part of field only.

*Left eye.* Tension normal. V as in right, or slightly better.

*Ophth. Exam.*—Extensive changes of an atrophic nature in discs, retina, and choroid: the latter in peculiar broad lines. Also dots and streaks at macula like albuminuric retinitis.

*Treatment.*—Strychnine and phosphorus pill. Continuous current daily.

June 27. *Right.* V = Fingers at 2 feet.
*Left.* V = Fingers at 4 feet.

August 13. V as on last report. Discharged.

CASE XVII.—*Recent rapid loss of sight. ? Locomotor ataxy. No improvement.*

Ann M——, married, æt. 60. Admitted October 28, 1879, under the care of Mr. Wordsworth. She had two miscarriages about ten years ago, but has had no family. Her health has been good until ten months ago, when she had severe headaches. These lasted for a month, and at the end of that time she noticed that the sight of her left eye had failed greatly. It has since got still worse, and about five months ago the right began to grow dim. In the last two or three months she has been much as now. She has pain in the lumbar region and sometimes has a feeling of constriction round the waist. Stands well in darkness.

*Right eye.*—Pupil dilated from atropine. Tension normal. V = Nil, or barest perception of the gas-flash.

*Left eye.*—Pupil inactive. Tension normal. No perception of light.

*Ophth. Exam.—Right.* Deep saucering of disc; veins rather tortuous; capillary circulation of nerve fairly good. Macula not marked by the usual difference of tint.

*Left.* Much as in right, but the outer side of the disc is here atrophic and white.

*Treatment.*—Continuous current daily.

She only gets a very faint light-impression with 25 cells.

November 6. Discharged. V not improved.

CASE XVIII.—*Recent failure of sight. History of Syphilis and of injury to spine. No improvement.*

Thomas R——, sailor, æt. 35. Admitted October 30, 1879, under the care of Mr. Lawson. He had syphilis twenty years ago. About ten years ago he was struck severely on the lower part of the back by the fall of a plank of wood: his back has been more or less painful ever since.

Four months ago "everything looked double;" the right eye was then drawn in towards the nose. A week or two later his sight became dim.

He suffers now from very severe headaches, with giddiness. Tendon-reflex low.

Pupils semi-dilated and feebly active. Tension normal.

*Right.* V = Fingers barely at 4″.

*Left.* V = Same as with right—rather clearer perhaps.

*Ophth. Exam.—Right.* Disc atrophic. Edge indefinite from marginal choroidal changes. Several spots of choroidal atrophy near disc.

*Left.* Disc pale and slightly swollen. Choroidal changes as in right.

*Treatment.*—Continuous current daily.

November 3.—Severe frontal headache, chiefly on right side. Relieved by Heurteloup's leech.

November 6.—Ordered dry cupping (with Heurteloup's cylinder) behind each ear. Iodide of potassium.

November 10.—Discharged. V not improved.

Of the above eighteen cases, six distinctly improved under treatment; four others received doubtful benefit; and eight did not improve at all, or grew worse. Of the six improved cases, two returned to hospital some time after their first discharge with their sight as bad as ever, and on this second occasion neither benefited by any treatment.

From the small number of cases, and from the fact that no such reliance was placed in the treatment by the electrical current as to justify one in trying it *alone*, no really conclusive results have been obtained. At the same time, when we consider the nature of the other treatment, and when we compare the above results with those obtained where no

galvanism was employed, a strong presumption must remain with us of the utility of the continuous current in certain cases. As regards the other treatment referred to, it consisted generally of nerve tonics (especially strychnine), iodide of potassium, and good feeding. The iodide I believe is utterly useless in cases such as the above. Strychnine subcutaneously administered has been strongly recommended, but our experience at Moorfields of its usefulness was most unsatisfactory in a series of cases in which it was tried by Mr. Buller, late house-surgeon. Possibly, however, strychnine, as well as other nerve tonics, and good food certainly, may be important adjuvants to the usefulness of the continuous current.

Notwithstanding the inconclusiveness of the results, however, it will be well to analyse the above cases with a view to ascertain how far it may be possible to prognosticate of any case as to the probable benefit to be derived from this mode of treatment. For this purpose I shall consider each group in reference to age, etiology, duration, and amount of amaurosis, colour-perception, occurrence or non-occurrence of pain during failure, and the ophthalmoscopic appearances. For the sake of brevity, I shall call the improved cases group A, the doubtfully improved $A^1$, and the unimproved cases group Z.

*Age.*—In groups A and $A^1$ it averaged thirty-one years, and in group Z thirty-four years. One of the oldest patients improved satisfactorily under treatment. Age *alone* would of course be useless as a means of prognosis but probably, other things being equal, the younger the patient the greater the probability of success.

*Etiology, &c.*—There was a history of a somewhat excessive use of tobacco, alcohol, or both, in one case of $A^1$ and of Z, but neither was to be confounded with a true case of toxic amblyopia.

There was a positive history of syphilis in two cases of A and in two cases of Z: and a doubtful history in one case of A and two cases of $A^1$.

There were symptoms of locomotor ataxy in two cases of A, in two cases of $A^1$, and in three cases of Z.

History of rheumatism in one case of A and in three cases of Z.

*Injury.*—In one case of $A^1$ and in four cases of Z.

*Renal Disease.*—In one case of $A^1$.

*Duration of Amaurosis.*—In group A it ranged from fourteen days to three years; in group $A^1$ from three months to three years; and in group Z from four months to four and a half years.

There is no doubt that the more recent the amaurosis the more likely it is to be benefited by any treatment, but a recent case may be otherwise an unfavourable one.

*Amount of Amaurosis.*—In group A, vision varied from fingers at 1 foot up to Jaeg. 4 with the better eye; in group $A^1$ from bare perception of large objects up to fingers at 5 feet; in group Z from bare perception of light up to Jaeg. 20. In one case of group Z the better eye saw Jaeg. 8, but as the failure was at least arrested for the time in this eye by treatment, it seems better to mention it separately.

The greater the amount of vision retained, therefore, the better hope of benefit. But here, as in the last series (*Duration*), the successful and unsuccessful cases overlap one another, showing that there must be some other very important element in the prognosis.

*Colour-Perception.*—Of groups A and $A^1$ only three were tested properly: all these were defective. Of group Z also three were tested: one was defective, two were good.

The unsuccessful cases, therefore, seem to have retained colour-perception better than the others, but unfortunately the majority of the cases were not properly examined in this particular.

*Occurrence of Pain.*—There were headaches more or less severe in five out of the ten cases of groups A and $A^1$, and in five out of the eight cases of group Z.

The fact of pain having occurred or not depends, of course,

on the true pathological history of the case; but in ignorance of the exact nature of the latter, I thought it well to make a distinct heading of such a prominent symptom. Its prognostic value seems nil.

*Ophthalmoscopic appearances.*—The large vessels of the disc were normal in size in three cases of groups A and $A^1$ but in no case of group Z. They were diminished in two cases of A and $A^1$ and in every case of Z. The size of these vessels was not noted in three cases of A and $A^1$ and in one case of Z.

The perivascular sheath was notably thickened in three cases of group A and in three cases of group Z.

There were visible retinal or choroidal changes in four cases of groups A and $A^1$, and in four cases of group Z.

There seems to be a definite connexion between the size of the large vessels and the probability of improvement under treatment, the prognosis being bad where the size is diminished.

As the result of this imperfect analysis, then, we would give the most favourable prognosis where the patient is young or middle-aged, with recent failure of sight and present ability to count fingers at least. Colour-perception is possibly defective. The disc is white, but the large vessels are of normal size. There is no history of severe injury to the brain or spinal cord, but perhaps there are symptoms of locomotor ataxy.*

## II.—*Retinitis Pigmentosa.*

While making the above investigation on the use of galvanism in optic atrophy, every such case that was

* For an excellent report on the use of galvanism in six cases of Optic Atrophy, see a paper by Mr. R. J. Pye-Smith, in Brit. Med. Jour. for May 18th, 1872. Of the six cases, two improved decidedly, and two doubtfully. It was at this gentleman's suggestion that I commenced this method of treatment.

Dr. Fraser, of Paisley, also reports a case in which there was decided improvement. See Glasgow Medical Journal, 1872, p. 163.

admitted as an in-patient was treated in this manner. Among the rest was the first case now to be detailed, in which the optic atrophy was secondary to, or at any rate accompanied by, retinitis pigmentosa. Here the result was so very encouraging and so much more brilliant than in ordinary cases of atrophy, that I determined to try the same treatment on the next case of retinitis pigmentosa that presented itself. Only one other such case was obtained, and here again the result was good.

For the other two cases given here I am indebted to Mr. G. A. Berry and to Dr. W. Charnley, who kindly undertook to try the effect of the same mode of treatment on any patients that might come under their notice.*

### Case I.

Watts T——, æt. 42, weaver, married. Admitted November, 20, 1878, under the care of Mr. Streatfeild. He has four children alive and three dead; the last two were stillborn. He had a sore under his foreskin about six years ago, but he gives no history of secondary symptoms. His sight, he says, has failed for the last eight years, and his health has been bad for about the same time. Lately he has been obliged to give up work. He is a thin man, with dark hair and complexion. Health delicate, with mental depression. He is unsteady when tested with his eyes closed, and he has a feeling of tightness round his waist. He says he feels "drunk and pulled down."

*Right.* V = Fingers at 3 feet.

*Left.* $V = \frac{20}{200}$; with $-36'' = \frac{20}{70}$.

Field contracted. Tension normal in both. Colour perception good, but sometimes confuses green and blue.

*Ophth. Exam.*—Well marked retinitis pigmentosa, with atrophy of disc and considerable diminution of vessels in each eye.

* These gentlemen continued the treatment steadily in their cases for a considerable time, and were most particular in mapping out the field at short intervals. The field-charts are very valuable records. It has been judged sufficient to publish here the field of each eye before commencing and at the conclusion of all treatment.

*Treatment.*—Iodide of potassium and the continuous current daily.

December 31.—*Right.* V = Fingers at 18 feet. $\frac{20}{200}$ and nearly $\frac{20}{100}$ in *extreme outer part of field.* With $-40'' = \frac{20}{100}$ centrally. His field has therefore improved.

*Left.* V = $\frac{20}{70}$; with $-40'' = \frac{20}{50}$ fairly.

January 28, 1879. *Right.* V = $\frac{20}{200}$; with $-40'' = \frac{20}{100}$.

*Left.* V = $\frac{20}{70}$ nearly, and with $-40'' = \frac{20}{70}$ and 'T' of $\frac{20}{50}$.

The light is bad to-day.

Iodide of potassium discontinued.

April 21. *Right.* V = $\frac{20}{70}$ barely, with $-40'' = \frac{20}{70}$. Reads Jaeg. 8.

*Left.* V = $\frac{20}{40}$ barely. Prefers weak concave, but sees no more. Reads Jaeg. 4.

Light fairly good to-day.

May 16. V as on last report. His general health has improved while in hospital. Discharged. He went home to Leicester, where Mr. Hodges kindly continued this treatment in the Ophthalmic Hospital there. Some months later he again came to Moorfields with his sight as good as on his discharge. (The notes of this visit cannot be found, and I only speak from memory).

### Case II.

James D——, æt. 33, printer, single. Admitted November 9, 1879, under the care of Mr. Tay. No consanguinity of parents. Maternal uncle was "short-sighted," but patient believes that he was able to see his way about at night.

Health has been good, except that he had very severe rheumatic fever when a boy.

He says he could never see so well as most people, but he

was able to do very fine work (reading theodolites, &c.) till lately. Twelve months ago his sight became so bad that he could not find his way about after dark, but he thinks that he has not got worse in this respect since then. He has never had any pain in or around his eyes. Recently he had to give up work on account of failure of sight, even by daylight.

He is rather thin. Hair and complexion dark: irides light coloured.

Pupils fairy active. Tension normal in both. Field of vision peripherally in each.

V = Jaeg. 4, barely, with each.

*Ophth. Exam.—Right.* Much pigment in retina in characteristic forms: it extends well up to the disc-margin. Slight choroidal atrophy near disc.

*Left.* Well marked choroidal atrophy at outer side of disc. Pigment not so much as in right, and not in such large masses. Retinal vessels considerably diminished in both eyes.

*Treatment.*—Continuous current daily. He uses twenty-five cells for about three minutes each morning.

November 15.—Immediately after the use of the battery to-day he can read Jaeg. 1, in a comparatively dull light.

December 20.—He was unwilling to admit improvement at first, but he has now no doubt of it. He reads Jaeg. 1 fluently with the right, and barely with the left eye: $V = \frac{20}{100}$ barely with each.

A few days ago he went back to his former employers and satisfied them that he was fit to return to his usual work. Discharged to-day.

CASE III.—*From Dr. Charnley's Notes.*

Fanny R——, æt. 20. Came to Moorfields as an out-patient October 6, 1880, under the care of Mr. Tweedy.

There is no history of consanguinity in marriage except that her grandfather married a second cousin. No insanity in the family, and no other member of it has weak sight.

General health has been fairly good.

Three years ago she first found a difficulty in walking alone in the street on account of running up against people: at that time she was suffering from amenorrhœa. Since then she has become nyctalopic.

She is a rather delicate looking blonde with light grey irides.

*Right eye.* $V = \frac{20}{100}$; with $-2 \cdot 0$ D $= \frac{20}{70}$. Jaeg. 1 at 12″.

*Left eye.* $V = \frac{20}{70}$; with $-1 \cdot 0$ D $= \frac{20}{40}$. Jaeg. 1 at 12″.

Field of vision very much restricted in each eye, not extending beyond the 10° circle in any direction. (See chart.)

*Ophth. Exam.—Right.* Disc seen only hazily as through a thin veil. At the lower part there is a tag of something like lymph in front of the vessels.

Arteries and veins of normal size. Near the disc, the fundus is beset with minute black specks: towards the periphery the pigment takes the usual stellate shape, and is in front of the retinal vessels. No streaks of pigment visible along the vessels. No perivasculitis. In the left eye the condition is very similar.

*Treatment.*—Continuous current daily. About 13 cells were used. The sponges were usually placed one on the closed eyelids and the other on the supra-orbital region on the same side, but both sponges were moved about along the skin within narrow limits. Occasionally the sponges were placed one on each eyeball.

October 7. Patient is sure she sees better. The chart of the left field was taken before and immediately after the use of the battery to-day: the former shows slight improvement from yesterday, the whole field being now a little beyond the 10° circle. The field after the battery is still wider, extending to the 20° circle downwards. The extreme periphery of both fields carefully tested now: it is found that in that part of the field she cannot distinguish a piece of white paper 5″ × 3″. The scotoma is therefore not simply annular.

Colour-perception good, but she confused a light sea-green with blue.

October 9. At extreme outer periphery of each field of vision she can now distinguish the hand against a dark back-ground, and can tell correctly the direction in which it is moved.

This small perceptive island is represented on the chart by an almost round space reaching from the 60° to the 70° circle in the left eye.

October 11. In both eyes a piece of white paper 5″ × 3″ is

seen well at the extreme outer periphery, but not between the limits 25° and 70°.

October 14. The central field is steadily enlarging and the peripheral island is increasing vertically.

October 20. *Right.* $V = \frac{20}{70}$; with $-2{\cdot}0$ D $= \frac{20}{30}$ nearly.

,, 28. *Right.* $V = \frac{20}{100}$; with $-1{\cdot}75$ D $= \frac{20}{30}$ partly.

*Left.* $V = \frac{20}{100}$; with $-2{\cdot}0$ D $= \frac{20}{30}$ partly.

From this time onward the field of vision of both eyes gradually widened. Improvement came latest above.

The treatment was continued daily, excepting Sundays, until December 18.

December 20. She was sent back into the country in the following condition:—

*Right.* $V = \frac{20}{70}$; with $-1{\cdot}50$ D $= \frac{20}{30}$ partly.

*Left.* $V = \frac{20}{70}$; with $-1{\cdot}50$ D $= \frac{20}{30}$ partly.

For field of vision now, see charts.

### Case IV.—*From Mr. Berry's Notes.*

Henry A——, æt. 22, admitted under the care of Mr. Wordsworth. Two first cousins have nyctalopia. The night blindness is one of his earliest recollections and must have existed, according to his mother's statements, when he was three years old. He does not think it any worse now than it ever has been.

He has been repeatedly treated by medical men in London and elsewhere, but observed no improvement from their treatment, which always consisted of *medicaments*. He is a short man; well-nourished and healthy looking. Complexion dark.

Reads Jaeg. 1 easily. V with $-3{\cdot}0$ D $= \frac{20}{40}$ barely.

His field of vision extends no further than 15° in either eye. He is completely red-green blind, and partially blue-yellow blind, so that there is almost total absence of colour-perception: of this he seems to have been aware. His power of distinguishing

shades varying slightly in intensity is diminished decidedly, though not to a great extent.

*Ophth. Exam.*—Discs of characteristic pallor. Vessels very decidedly diminished in calibre. Well marked retinal pigmentation coming close up to the papilla, but scanty towards the ex-periphery.

*Treatment.*—Continuous current daily. Number of cells used 19 to 21, and employed for about ten minutes.

Until the end of November the state of the field was ascertained every day, *i.e.*, one day the right, and next day the left eye. For the first half of this time the charts only indicate the amount of field standing after electrisation. In the second part it was always taken both before and after applying the current. The difference shows, I think, an improvement on the whole *immediately after* the current, though once or twice this was not apparent. At all events, at the end of the treatment the field was undoubtedly more extensive than at first. The improvement was more marked in the right eye, and only took place outwards and downwards, where it extended to from 70° to 90°. Although the field extended in this way, the peripheral vision was very imperfect and undecided.

There was no improvement in central vision, nor in the night blindness.

In this case, as in Dr. Charnley's, a perception-island appeared near the outer periphery of the field in each eye. This is first noted in the chart on November 26; at the same time the central field was prolonged in a tongue-shaped projection towards this island, until at last they met, giving rise to the peculiarly shaped field depicted in the chart of December 10.

On my writing him, enquiring how his sight was, and wishing him to come to hospital and be examined, he replied from the country, on January 20, 1881: "I am thankful to say that my sight is slowly getting better. My sight was getting better during the time I was in Moorfields Hospital."

All the cases of retinitis pigmentosa were therefore improved by this treatment.

On looking up the literature of the subject, I find that galvanism has been tried by several different authorities in this disease, but encouraging results are only reported by Neftel and Dor. The former reports one case*. Here the patient was unable to read, and could not walk alone in the street before treatment; after a continued use of galvanism he could walk alone and read for several hours daily.

Dor† gives three cases, in all of which there was considerable improvement in central vision. Why others should not have been equally successful may possibly be partly explained by their method of applying the current, or by the vision having been too far deteriorated before treatment was commenced.

My reasons for believing that galvanism will prove especially serviceable in the treatment of retinitis pigmentosa, are founded chiefly on the above experience of its use, and partly on a consideration of the nature of the failure of vision in these cases. The pigment deposited in the layers of the retina absorb the rays of light, which would otherwise penetrate and stimulate certain of the rods and cones. The nerve fibres in connexion with these retinal end-elements are now thrown absolutely out of employment. Corresponding with the distribution of the pigment over the fundus, in early cases we find an *annular scotoma;* but soon the more peripheral field also fails, the part within the blind ring contracts more and more, and finally central vision itself deteriorates. Night-blindness becomes a more prominent symptom, since much of the light is lost by absorption in the pathological pigment; the deficient blood-supply interferes with rapid tissue change, and a degenerated nerve conveys with difficulty stimuli which in themselves are abnormally feeble. The mere fact of the nerve being no longer acted on by normal stimuli assists in the atrophic

* Neftel, Galvanotherapeutics. *See* Nagel's Jahresbericht, ii, pp. 207–8.

† Dor. Archiv. f. Ophth. Bd. xix, Abth. 3, pp. 342–5.

process, and it is this secondary atrophy from disuse that I believe the continuous current is qualified to prevent.

From the experiments of Holmgren, and later of Dewar and McKendrick, we know that the stimulus of light on the retina causes an increase of the normal electrical current passing along the optic nerve and tract from periphery to centre. Again, as a matter of personal observation, we know that the continuous current applied to the temples or over the eyes causes a sensation of light. From these facts we may, without assuming the identity of the electrical and nervous currents, argue that the optic nerve conveys to its centre in the brain, both the electrical current and the current originated in the impression produced upon its end-organs by waves of light. It is this *conductivity* of the nerve which the continuous current is capable of stimulating and of preserving.

Possibly galvanism has also a *temporary* effect in dilating the retinal blood-vessels, and thereby improving the nutrition of the tissue. Such an effect has been observed by some authorities (Roosa, Lornig, Hackley) from its use in the normal eye. In the cases I have seen, there has certainly been no *permanent* result of this kind observable after long-continued treatment.

But whether the above explanation be the true one or not, I trust others will be induced to try this mode of treatment. Were it only to prove serviceable in exceptional cases, it would still be our duty to *try* it in every case, until we knew precisely in what class it might be expected to do good.

Galvanism has, in many departments of medicine, proved very disappointing; this fact, coupled with its frequent association with charlatanism, has caused a professional prejudice against its use. This will deter no one from employing it where he has reason to expect benefit. Again, it may be argued that cases such as the above only improve while under treatment, and that when the patient leaves off attendance, his condition deteriorates as before. This argument will apply to many other forms of treatment quite as forcibly as to galvanism, even supposing it be always true.

There is, moreover, nothing to prevent even a hospital patient learning to use the battery for himself; and the cost of an efficient instrument now places it within the reach of, at any rate, *most* of those in whose cases it would be valuable.

I may add a word of caution to any one using galvanism for the first time in any case, especially when it is to be applied to the head. Always begin with a weak current (of five or seven cells), and let the first application be a short one. When the idiosyncrasy of the patient is thus determined, the current may be gradually increased if necessary, and the application continued for a longer time.

I cannot conclude without thanking the surgeons to whom the above cases belonged for their kindness in allowing this method of treatment to be tried, and for their courtesy in permitting me to publish the results.

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## EXPLANATION OF FIGURES.

FIG. 1. Chart of visual field of right eye in Case 3, p. 184. In this and in the other figures the central blank space represents the entire field *before* treatment; while the shaded portion represents the *improvement*; the remaining (dark) portion is the blind area.

FIG. 2. Visual field of left eye in above case.

FIG. 3. Field of right eye in Case 4, p. 186.

FIG. 4. Field of left eye in same case.

Fig. 1.

Fig. 2.

Fig. 3.

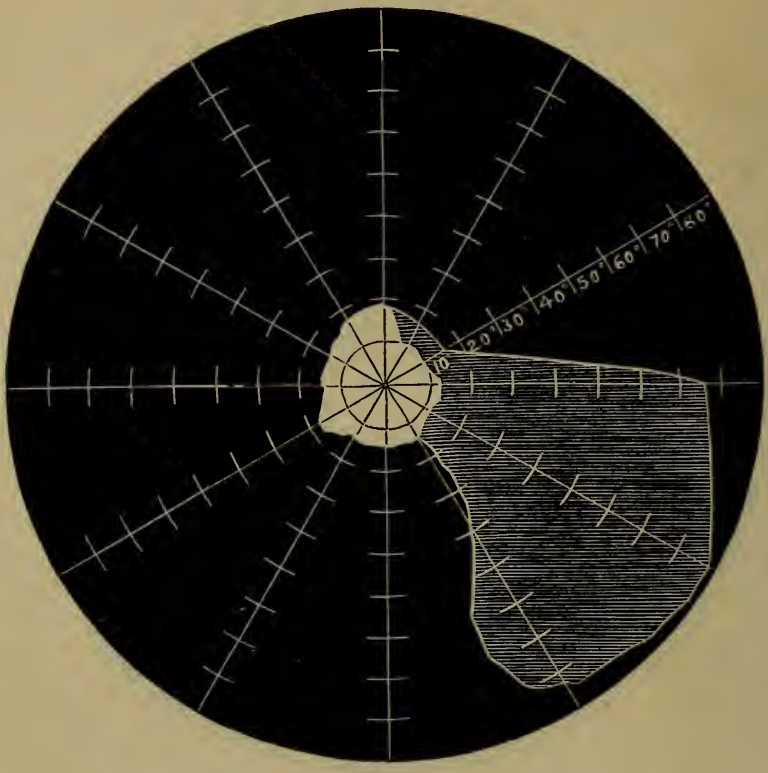
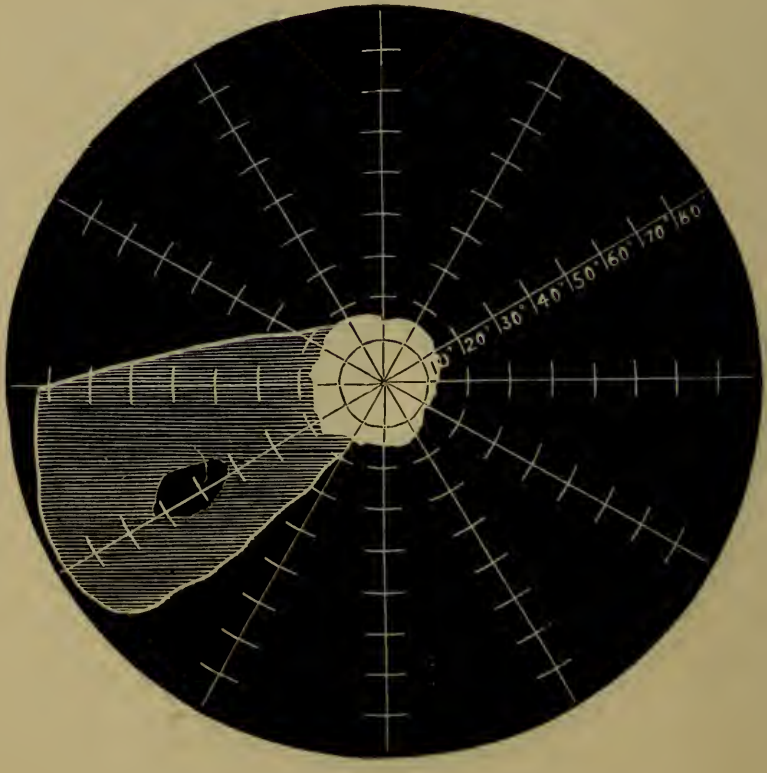



Fig. 4.



## THREE CASES OF NEW FORMATION OF BLOOD-VESSELS IN THE VITREOUS.

By W. Charnley, M.A., M.D. (Cantab.); and L. Webster Fox, M.D. (Jeff. Med. Coll., U.S.A.),
*Clinical Assistants at the Hospital.*

The presence of newly-formed vessels, visible with the Ophthalmoscope, in an otherwise fairly healthy vitreous, is a somewhat rare phenomenon; it is remarkable that the three following cases should have been under observation at Moorfields at the same time.

Case 1 was that of a young girl, E. C——, æt. 14. She had squinted inwards with the right eye since early childhood, and had had a tenotomy of the right external rectus done. A year ago she noticed a black thread in front of the right eye. There had never been a blow on this eye, and there was no suspicion of syphilis. Health always good. Menses began at the age of 13, and continued regularly.

She first came to hospital in May, 1880; then in each eye V was equal to $\frac{20}{50}$.

Sphero-cylinders slightly improved her sight, and were accordingly ordered.

She was not seen again till August, 1880, and then the following note was taken:

*Right Eye.* The margins of the disc are decidedly hazy; vessels enlarged and slightly tortuous; difference between veins and arteries ill-marked. Below the disc is what at first sight appears to be a detachment of the retina, small vessels being seen projecting forwards into the vitreous. On closer examination it is seen that what are undoubtedly true retinal vessels lie a considerable distance directly behind the above-mentioned vessels; so the latter cannot be detached retinal vessels; they are probably vessels of new formation sprouting into the vitreous. They can be traced backwards to the lower margin of the disc; anteriorly they form a succession of

loops (Fig. 1), and seem to terminate near a glistening white mass in the vitreous. The most anterior part of these growths can be seen with + 5 D, the fundus being nearly emmetropic.

Upwards and outwards is seen a white band of connective tissue, crossing the fundus obliquely; it arises from the upper part of the fundus at a point (beyond the limits of the illustration) where it is in contact with the retina, and partly constricts a retinal artery. As it proceeds down and out it lies upon a level a little more anterior; so that it passes slightly in front of a large retinal vessel, which on the distal side of the point where it is crossed becomes much larger and tortuous. A little further down and out this retinal vessel and the band have again come into connexion through the sudden bending down of the former, and at this point five vessels are seen to arise and pass forwards into the vitreous. It cannot be certainly said that they arise from the large retinal vessel, but they certainly appear to do so. A very fine supporting framework of connective tissue seems to accompany them into the vitreous, where they form loops as shown in the drawing (Fig. 1). A largish retinal vessel can be seen some distance directly behind them. Near some of these fine vessels, but without demonstrable connexion with them, are two or three half-transparent round or oval discs, having perhaps one-sixth the diameter of the optic disc. The nature of these is not clear; probably they belong to the framework just mentioned. The most anterior of these vessels just described can be seen with + 6 D. In the neighbourhood of the macula are some traces of old choroiditis.

*Left Eye.* Fundus emmetropic; margins of disc hazy; veins slightly swollen; summit of disc seen with + 2 D.

The patient was ordered five grains of iodide of potassium three times a day, and to this, after a couple of months, was added a drachm of the solution of the perchloride of mercury. Later moderate doses of calomel and opium were tried, but had to be discontinued, and the former medicine resumed.

The only change at the present time (February, 1881)

after five months of treatment, is that in the right eye the vitreous is much clearer, and the newly-formed vessels proceeding downwards and outwards from the disc appear to have shrunk considerably; in left eye there is no apparent change.

V in each eye is still $\frac{20}{50}$.

The following are the notes of our second case:—

Maud J——, æt. 19. Draper's assistant. First attended as an out-patient in April, 1880, complaining of "dimness of vision." Family history irrelevant. History of iritis dating four months prior to attendance. Before this attack the patient always had good sight and enjoyed good health. She attributed the inflammation of her eyes to catching cold during a menstrual period. No history of syphilis could be elicited.

Right Eye. $V = \frac{5}{200}$. Left Eye. $V = \frac{20}{100}$.

No improvement with glasses.

*Right Eye.* The cornea was hazy with well marked Keratitis punctata, especially in its lower part. In the lower part of the anterior chamber there was a small deposit of organized lymph, on which a few delicate blood-vessels could be seen by oblique illumination. Colour of iris unaltered. There was almost complete posterior synechia; a small segment of the upper and outer quadrant of the iris responding to instillations of atropia. No reflex from the fundus obtainable. Tension normal.

*Left Eye.* Cornea clear. A few spots in the lower half of the cornea on Descemet's membrane. Colour of iris unaltered. Dilated fully with atropia. Lens clear. The vitreous was hazy, especially in its posterior part; and this haze was most marked in the neighbourhood of the disc. The retinal vessels, so far as they could be seen, were normal. The outlines of the disc could not be defined, but the central vessels were indistinctly visible. A floating vessel of a pale red colour and peculiar spiral course, about one-third the diameter of the central artery, could be traced forward to near the equator bulbi, then turning upwards and outwards was lost in the vitreous. Neither its point of

origin nor ending could be ascertained owing to the haziness of the media.

Tension normal.

The patient was ordered two grains of mercurial pill twice daily, and the pharmacopœial solution of sulphate of atropine three times a day to the right eye. This treatment was continued for two weeks, when salivation was produced. The number was then reduced to one pill daily, and a gargle of chlorate of potash was given.

At this time there was no improvement of vision; and the objective signs were unchanged. This treatment was persisted in for three months, at the end of which time the vitreous was sufficiently clear to enable a drawing to be obtained, as represented by Fig 2. The largest vessel had its origin from the disc. It came straight forward into the vitreous, and then curved upwards, and could be traced to a point in the retina, where an artery crossed a vein. This point was situated in the equator bulbi above and outwards. The group of smaller vessels sprang from the centre of the disc and they were suspended in the vitreous. The majority of them formed at their anterior extremities a close spiral coil, and then retraced their course to their place of origin.

While under treatment the calibre of these vessels gradually lessened, and by the beginning of December (eight months after the date of admission of the patient) they had entirely disappeared.

During the last three months, and while the patient was taking the mercurial pill daily, five small growths made their appearance on different parts of the iris of the right eye. They were situated for the most part midway between the pupillary and ciliary margins. They were of a greyish-brown colour, grew to the size of a millet seed, and then gradually disappeared.

On January 25, 1881, the following notes were made :—

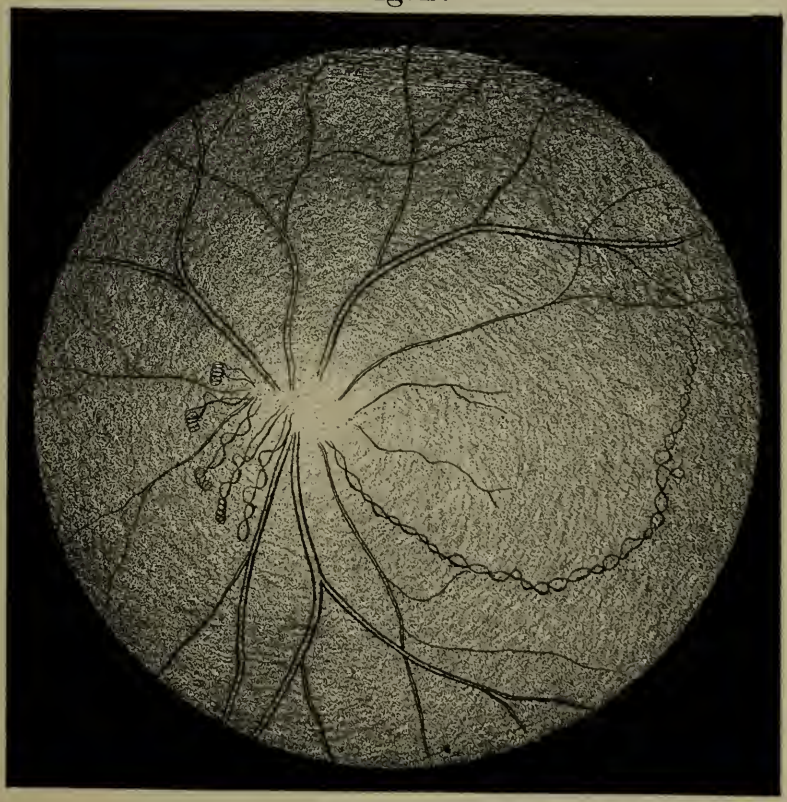
*Right Eye.* $V = \frac{5.}{200}$ Cornea hazy. Keratitis punctata and posterior synechia unchanged. No reflex from the fundus obtainable.

*Left Eye.* $V = \frac{20}{20.}$ The few punctate deposits of Descemet's

*Charnley & Fox, New vessels in the Vitreous.*

Fig. 1.

Fig. 2.



membrane unchanged. The media were perfectly clear, so that a complete view of the interior of the eye was obtainable. The fundus presented nothing abnormal, and no traces of the new vessels described were observable.

The third case was brought to our notice by Dr. W. A. Fitzgerald.

The patient was a woman, æt. 51. There was no specific history. She had just ceased to menstruate.

She first came to the hospital in April, 1880, with clots in the right vitreous.

V then was R. E. = $\frac{20}{200}$, L. E. = $\frac{20}{30}$.

She attended from time to time, taking tonics, till December, 1880, when

V was R. E. $\frac{20}{30}$, and with + 3·0 D Jaeg. 1.

L. E. $\frac{20}{70}$, and Jaeg. 16.

On ophthalmoscopic examination of the right eye the appearances were as recorded in the following note:—

The vitreous is slightly hazy. There is no detachment of the retina. Downwards from the disc is a small greyish spot of connective tissue on the retina, from which a bunch of vessels extends upwards and forwards into the vitreous. These vessels correspond exactly with the description given of those in Fig. 72 of Jäger's Atlas. Some present free extremities, while others form closed loops, returning to their place of origin, after having at their most anterior part formed a very fine and close spiral. They float freely in the vitreous, moving with the motions of the eye.

The fundus presents nothing else abnormal except that at the disc, near its lower margin, is a small tag of apparently connective tissue. In the left eye are numerous clots of blood in the vitreous, and no clear view of the fundus is possible. In both eyes tension is normal. Two months later the right vitreous was so hazy that the fundus could not be seen, and on this side V was less than $\frac{20}{200}$.

In left eye at the same date V was $\frac{20}{50}$, and with a concave lens of 1·0 D $\frac{20}{20}$.

There was no albuminuria: no cardiac murmur. The vessels were not specially atheromatous.

We have been able to find only four cases of new formation of vessels in the vitreous visible with the ophthalmoscope hitherto recorded. Mauthner, in an admirable article on this subject,* quotes two. He insists upon only those cases being included in this category, in which the new formation of vessels is the prominent symptom. One of the cases he records was described by Coccius in 1859, and seems to correspond pretty closely with Case 1 of this paper. A full account of the second case, with a plate, is to be found in Jäger's Atlas, Pl. xv, Fig. 72.

This resembles very closely our second case. In both there was the same curiously convoluted bunches of vessels, which Mauthner aptly compares to the microscopical appearance of an injected Malpighian body; and in both the abnormal vessels completely vanished under mercury with complete restoration of vision. In our case there were in addition to the vessels described by Professor Jäger, the large vessel of new formation above referred to, and the curious growths on the iris of the other eye.

Schweigger, in some remarks on this subject,† says that new formation of vessels in the vitreous in eyes, which are blind from choroiditis is by no means uncommon; but, as in these cases the lens and vitreous have generally lost their transparency, such formation can seldom be seen. The cases that Mauthner describes he thinks of a totally different nature; he ascribes them to retinitis, and regards the hyperæmia and loss of transparency of the retina as sufficient evidence of this retinitis. References to two other cases of a

* Lehrbuch der Opthalmoscopie, p. 114.

† Handbuch der Augenheilkunde, p. 417.

similar nature are given by him. These will be found fully described in the "Bericht der Wiener Augenklinik," 1867, pp. 106, 114. In one of these the eye was removed and the connexion of the newly formed vessels with those of the retina demonstrated.

It is worthy of remark that two of the cases described in this paper simulated very closely a detached retina. The diagnostic point, of course, was that some distance directly behind the abnormally situated vessels undoubted retinal vessels could be seen in their natural situation.

It is possible that the true state of things is sometimes mistaken, and cases of new vessels in the vitreous regarded as cases of detached retina. We are indebted to Messrs. Tweedy, Streatfeild, and Tay, to whom these cases respectively belong, for permission to publish them.

Since the above article was written, a case similar to the above has been published in the "Centralblatt für Praktische Augenheilkunde," for February, 1881. It will be found on p. 49, with a sketch.

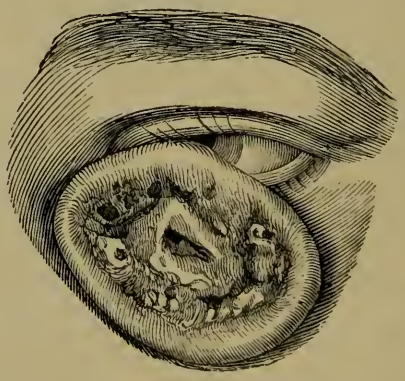
## A CASE OF PRIMARY EPITHELIOMA OF THE LOWER EYELID.

By George Lawson, F.R.C.S.

*Surgeon to the Royal London Ophthalmic Hospital, and to the Middlesex Hospital.*

The following case is an example of a very rare affection, a primary epithelioma of the eyelid. Epithelioma is occasionally developed from a rodent ulcer in the vicinity of the eyelid, but it is seldom that it attacks the eyelid as a primary disease.

Case.—Thomas B ——, æt. 65, was admitted into the Royal London Ophthalmic Hospital on November 23, 1880, with a primary epithelioma of the left lower eyelid. He said that it began as a small pimple about three months ago; it ulcerated and rapidly increased until it attained its present dimensions, and it was still growing. The ulcer was of the size, and presented the appearance, which is well shown in the woodcut. It in-



volved the greater portion of the margin of the lower lid; it was a distinctly new growth, very solid when felt between the fingers, and could be raised from the subjacent tissues. The margin of the ulcer was hard and rounded, and the whole surface of the growth presented a coarse, ulcerated surface, covered in parts with a thick yellow purulent secretion. There were no enlarged glands to be detected. As there was no doubt about the nature of the growth, I completely excised it, and left an open wound to granulate and cicatrize. The patient did well,

and left the Hospital in November 23, with the wound very nearly healed. The patient has since continued well.

*Remarks.*—Rodent ulcer and epithelioma are undoubtedly closely allied affections, although rodent ulcer differs in some important respects from epithelioma.

Rodent ulcer does not affect lymphatic glands, whilst epithelioma does, and often at an early stage of the disease.

Rodent ulcer is a rather dry ulceration with only little secretion and no fœtor, and the granulations are small. In epithelioma the secretion from the ulcerated surface is abundant and fœtid, and the granulations large, exuberant, and often in bosses.

Rodent ulcer is confined to the upper part of the face, whilst epithelioma, although it has a preference for certain localities, may under certain conditions attack any part of the body.

The points of resemblance between rodent ulcer and epithelioma are :—

Both rodent ulcer and epithelioma are new growths, composed mainly of epithelial cells ; and the new growth is the only part involved in the ulceration. As the growth increases and includes more of the skin and deeper tissues, so the ulceration continues to extend ; but the ulceration does not go beyond the new deposit.

On one point I am quite settled, that rodent ulcer, if left, will in time so change its character as to become true epithelioma. I cannot say whether this change is due to a mere progress of the disease, or whether it is that rodent ulcer is peculiarly apt to have superadded to it the characters of epithelioma after the same manner that old standing ulcers, unhealed wounds or scars, or other simple sores, become epitheliomatous.

The following case is an example of a frequently recurring rodent ulcer ultimately becoming a true epithelioma.

I had a female patient in the Middlesex Hospital from whom I removed a typical rodent ulcer beneath the lower eyelid,

The patient soon recovered and left the hospital. It recurred thrice in the space of the next five or six years, and on each occasion I excised it completely. About two years after the last excision, the patient again came back to the hospital, but this time with well marked epithelioma in the same locality. The disease had completely changed its character, it was no longer rodent ulcer, but a typical example of epithelioma.

## VOLUNTARY NYSTAGMUS.

By George Lawson, F.R.C.S.

*Surgeon to the Royal London Ophthalmic Hospital, and to the Middlesex Hospital.*

A short time since a gentleman, after he had consulted me about himself, told me that he possessed the power of shaking both his eyes, and that he had been able all his life to shake them at will. At my request he gave me a performance. First, making both his eyes steady, he then set both into rapid lateral motion, so rapid that the outline of the cornea was completely lost to view. The movements were really an exaggeration of what is seen in lateral nystagmus, but they were so rapid that the margins of the cornea could not be defined.

I have seen another case of voluntary nystagmus in a gentleman who was formerly a House Surgeon in the Royal London Ophthalmic Hospital, but the lateral movements of the eyes were not nearly so rapid as in the case above related. Before setting the eyes in lateral motion, he had to converge them sufficiently to squint, and then he could simulate lateral nystagmus. The movements were completely under his control, and could be started and arrested *instanter*.

## A CASE OF SIMULTANEOUS SUB-CONJUNCTIVAL DISLOCATION OF BOTH CRYSTALLINE LENSES, CAUSED BY THE KICK OF A HORSE.

By J. C. Wordsworth, F.R.C.S.

*Senior Surgeon to the Royal London Ophthalmic Hospital.*

G. W., æt. 68, an agricultural labourer, was admitted in July, 1880. Eight weeks previous to admission, he had received a kick from a horse on his face, by which he was knocked down, and rendered unconscious for a few minutes. The bones of his nose were fractured, and both eyes were ruptured.

At the time of his admission into the hospital, each crystalline lens was lying under the conjunctiva at short distances from the cornea, a little above and to the inner side of its margin; having escaped through a laceration in the scleral coat of each eye, concentric with the upper and inner edge of the cornea. There was a good anterior chamber in each eye; the corneæ were clear, pupils drawn upwards and occupied by dark clots; and the eyes were almost free from pain. In two weeks after admission (during which a drawing had been made of the eyes), he had so far recovered from the injury that the lenses were removed by division of the conjunctiva over them. Little or no irritation followed, and he left the hospital in a few days.

In December he was re-admitted to be examined for glasses, when his vision was as follows :—

R. E. with 10.50 D. V $\frac{20}{20}$ and with 15 D V = Jaeg. 1 at 7″.

L. E. with 10.50 D. V $= \frac{20}{40}$ and with 15 D V = Jaeg. 1, slowly.

In each pupillary space there were still some remains of blood-clots.

## A CASE OF GLAUCOMA PRECEDED BY OPTIC NEURITIS, WITH REMARKS.

By L. Webster Fox, M.D., and W. A. Brailey, M.D.

Emma W——, æt. 28. Under care of Mr. Streatfeild. Twelve weeks before attending the hospital she had severe pain in the right frontal region. Two days later, on awakening, she fancied she saw something moving in front of her right eye, and on closing the left, she found that only perception of light remained.

On admission as in-patient, her condition was as follows: left eye H = 1 D. Vision and accommodation normal. Fundus normal. Right eye, no perception of light; cornea clear; good anterior chamber; pupil semi-dilated and sluggish: optic disc scarcely recognizable owing to extreme swelling of it and the surrounding retina; the veins were enormously enlarged and tortuous, and of an inky-black colour; the arteries were about normal. There were numerous large irregular hæmorrhages scattered extensively over the fundus. The neighbourhood of macula was white and opaque. Tension normal. General condition: the patient was a well-developed healthy looking woman. Menstruation regular. No history of rheumatism nor any severe illness. Heart sounds normal. Urine sp. g. 1018; no albumen nor sugar. Her father had had two slight attacks of gout in the great toe, but there was no history of any other hereditary complaint. She remained an in-patient for 18 days, during which time she was treated with mercury and iodide of potassium. She was discharged in the same condition as on admission. During her stay in the hospital she had no pain, and tension continued normal.

On December 24, 1880, she returned. Her eye had become painful; the tension was increased; there was a good anterior chamber; pupil semi-dilated and fixed. One week later the tension was greatly increased (T 3). Indeed there were all the signs of acute glaucoma. No details of fundus were visible.

She was again admitted as in-patient, and treated with instillations of eserine, under which the pupil became semi-

contracted, the tension fell, and the pain was slightly relieved; but after a few days the tension increased again (T 3) and the pain became very severe. Sclerotomy was performed; this reduced the tension, and relieved the pain for a short time. After the lapse of twelve days, tension being again + 3, and the pain very severe, the eye was excised.

The microscopical appearances were generally normal, except that the vitreous contained a few hæmorrhages in its peripheral part. The lens was *in situ*. Microscopical appearances: optic nerve, many large nuclei in its trabecular tissue, more numerous near the eye than farther back. The central vein and artery imbedded in and obscured by a great accumulation of deeply staining nuclei. Only one vessel, probably the artery, discernible; lumen small; walls thickened and much infiltrated with nuclei. Farther back from the eye both artery and vein recognizable and patent, with the walls thickened and slightly inflamed; more so in the case of the vein than the artery. The optic disc was markedly swollen, and the lamina cribrosa pushed back. The retinal veins large, varicose, and enormously distended with blood. The arteries not above their normal size, and of uniform appearance. Enormous numbers of blood corpuscles extravasated, especially in the nerve cell and nerve fibre layers of the retina. The choroid not inflamed, though its veins were distended with blood. The ciliary body: muscular fibres inflamed and atrophied to little more than one-half their normal bulk; arteries enlarged. The ciliary folds atrophied and widely removed from the iris.

The iris atrophied, and the peripheral part adherent to the cornea. A slight excess of nuclei in the cornea. The sclerotic: a slight infiltration of nuclei more marked in the episcleral tissue, and in the walls of its blood-vessels. One enlarged vessel in its course through the sclerotic to the ciliary muscle thickened, surrounded, and blocked by an enormous excess of deeply staining nuclei, quite undistinguishable from those of its walls, apparently a thrombosed and dilated anterior ciliary vein.

*Remarks.*—The following questions suggest themselves:—

Did this disease start as an inflammation or as a thrombosis? The ophthalmoscopic appearances speak for the

latter, and also, though not very strongly, the suddenly discovered loss of sight.

Against it, and in favour of inflammation, is the precedent pain and, perhaps also, the fact that there is evidence of the independent thrombosis of two veins at points comparatively distant from each other. But if it began as an inflammation causing thrombosis, as, indeed, seems more probable, was it primarily of the optic nerve, extending thence to the ciliary body, or were both invaded simultaneously? We rather incline to the former of these two views.

In our opinion a neuritis caused an acute inflammation of the walls of the central vessels with a resulting thrombosis of the vein. The inflammation then extended forward, especially along the loose episcleral tissue and its blood-vessels, where it found increased expression in the walls of its numerous veins. Passing inwards along the anterior ciliary vein, it set up acute inflammation in the fibres of the ciliary muscle. The enlargement of the arteries of the ciliary body may also be due to the venous thrombosis.

The general condition of the eye, it will be observed, is that of an acute glaucomatous inflammation.

For cases similar in some respects, see the last number of these Reports, Vol. x, page 94, case 395, and Vol. viii, page 9.

Cases in which an embolism of the central artery of the optic nerve of an eye previously healthy has been followed by increased tension have been met with at Moorfields.

## CASE OF CYST (CHOLESTEATOMATOUS) OF THE IRIS.

By Simeon Snell,

*Ophthalmic Surgeon to the Sheffield General Infirmary, and to the Institution for the Blind.*

Frederick C—— æt. 20, from Hoyland, near Barnsley, was admitted into the Sheffield General Infirmary under my care on December 7, 1877.

Eleven months previously he was engaged chiseling a boiler plate when a piece of the metal flew off and struck his left eye. He noticed the particle fall again to the ground. At the time of his admission an opaque line was observed running obliquely across the cornea from its upper and outer margin to rather above its centre. Just behind and somewhat below this scar, in the anterior chamber and on the surface of the iris, was a glistening pearly white elevation; it was about the size of a pea, or perhaps larger, but more oval in form, extending from the periphery of the iris almost to its free margin. It seemed, especially at its external portion, to be in contact, or nearly so, with the posterior corneal surface. The lens was opaque. V = shadows. It would appear that a few days after the accident he could see fingers, and that afterwards his vision improved. Four months before admission, however, sight became worse, and it was then that his attention was drawn to the peculiar appearance on the iris; it was, however, very small at that time.

The right eye was in no way affected.

*Operation.*—December 10. Under ether the cyst was removed. A broad needle bent at an angle was introduced simultaneously with each hand on each side of the tumour, and on the needles being withdrawn, a Weber's canaliculus knife was passed into the lower opening and gently insinuated between the cyst and the cornea, and being brought out again at the upper incision, the bridge of cornea between the two openings was cut across by it. The piece of iris with the cyst upon it was next seized with forceps and excised. The cyst was removed entire.

Recovery was good, and on January 18th, 1878, it is noted that the traumatic cataract was operated on by the suction curette.

A good deal of opaque capsule resulted after this procedure, and on two occasions was torn through with a needle, some benefit accruing. Subsequently, however, another iridectomy was performed by the side of the former one, on account of the pupil still remaining plugged by capsular opacites. This improved vision, and on June 3rd, 1879, he could read Jaeg. 16 with a + $2\frac{1}{2}$-inch lens.

My friend Dr. Dyson, Physician to the Sheffield Public Hospital, was present at the operation, and kindly undertook to examine with me the cyst, and he made the following report:—

"The cyst when examined more carefully was found to be of a greyish-white colour and had a semi-transparent wall. Though entire, when removed it burst on being transferred to a bottle containing glycerine. Two separate portions were distinguishable; one (probably the anterior wall) was semi-transparent and had some of the contents of the cyst attached to it; the other had a more fibrous and membraneous aspect and was attached to that portion of the iris which was removed by the operation.

"A microscopical examination showed that the *contents* of the cyst consisted of a number of very large clear closely packed cells, like fat-cells, in which no nucleus was discernible; of cholesterine crystals in great numbers; of a large quantity of fatty matter both in the purely granular and oily forms; of pigment cells and granules in small quantity; of sparsely scattered tesselated epithelium and of a homogeneous purely granular material. The delicate anterior wall consisted of homogeneous material pervaded by very fine moderately well defined fibres. The membraneous-looking portion attached to the iris was associated with fatty materials and *débris* similar to those forming the contents; but it consisted mainly of fibres which were more sharply defined, larger in size, and more systematically arranged in bundles than those found in the anterior wall."

*Remarks.*—Cysts of the iris are of sufficient rarity to put on record the foregoing case, with the description of its structure, and, moreover, the method of operating is of interest. The great majority of cysts are associated with injury. Rothmund collected 37 cases, all he could find recorded, and of these there was a history of injury in 28. Hulke, also, in Vol. VI of these Reports, has described the structure of these

little tumours, and tabulates a series of published cases. He says "in fifteen out of nineteen cases which I have been able to collect, as also in the two now first recorded by myself, as well as that published in this number by Mr. Wordsworth, there was a distinct history of foregoing mechanical injury." After a search, although the period does not thoroughly embrace that which has elapsed since Mr. Hulke's cases were published, I have found 13 additional cases recorded, or including my own, now noted, 14. Out of this number 10 are distinctly described as associated with injury, whilst three are stated to be without "a history of injury," and one is mentioned as having "no cause able to be ascertained."

Removal of the entire cyst, if practicable, appears the correct method of treatment, especially in one of the character described. Needling or removing a portion of the wall of the cyst, as advocated by several authorities, would seem to be more applicable to the serous variety. The mode of operation adopted in the case I have recorded is one which will prove of service in the removal of growths in the anterior chamber, encroaching on the inner corneal surface, and in the present instance almost any other procedure would have been attended with rupture of the cyst. Mr. Carter describes and advocates an almost similar method in his work on "Eye Diseases," and relates a case of sarcoma of iris he successfully removed by it.

## SOME CASES OF DIPHTHERITIC CONJUNCTIVITIS.

By James E. Adams, F.R.C.S.,

*Assistant Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the London Hospital.*

During the last few years a very considerable number of cases of membranous conjunctivitis have come under observation at the Royal London Ophthalmic Hospital, most of them being of only a moderate degree of severity. They are for the most part characterised by a moderate amount of swelling of lids and of the ocular and palpebral conjunctiva, frequently there is no chemosis at all, and when present it is usually slight. There is a thin opalescent pellicle on the palpebral portion, most marked in the upper lid, uniform in thickness, easily peeled or rubbed off, and leaving a slightly granular or bleeding surface beneath. Frequently only one eye is affected; there is often a history of throat affection in other members of the family or amongst neighbours, though sometimes the most careful enquiry fails to elicit any such facts. The cornea usually escapes altogether, and the disease subsides in from ten days to a fortnight. The usual plan of treatment adopted at Moorfields is frequent and liberal washing with a lotion of sulphate of quinine, the membrane being pulled off as often as it is found to be present at the time of washing. Under this treatment nearly all cases of this moderate degree of severity get well, and apparently this plan is all that can be desired.

Two cases, however, have occurred lately in my practice which have forced the conviction upon me that when the disease occurs in its typical form our quinine lotion is utterly useless, as, indeed, are all other applications that have been tried hitherto.

The first of these two cases was, if possible, the more severe of the two, and reminded one very forcibly of the

graphic description of this disease by our late colleague, Soelberg Wells, a description taken from cases occurring in continental towns where, as is well known, this disease does occur in all its virulence.

The patient, Thomas Donovan, æt. eleven months, was brought to me on the fourth day of the disease. The upper eyelid was immensely swollen, hard, brawny, and the conjunctival surface everted and projecting below the lower one. Its conjunctival surface was not covered by a distinct pellicle, but was mottled with dirty yellow patches interspersed with extravasation, and the only false membrane visible was a little piece in the retro-tarsal fold at the outer angle. The lower lid was affected in a much less degree, there was considerable chemosis, the cornea was hazy and beginning to ulcerate in the centre, and the whole globe was prominent. The extreme tension of the cutaneous surface of the upper lid combined with proptosis gave rise to a suspicion that there might be an abscess, and an exploratory puncture was made deep into the orbit, but no pus escaped. All doubt as to the diagnosis was removed the next day, by the fact of the epithelium of the cutaneous surface of the upper lid peeling off, leaving a surface as though a blister had been applied, and this became covered with a typical false membrane, which was persistently renewed for many days after each removal. This patient was seen daily and treated with the quinine lotion diligently, but no signs of diminution of the disease were visible for ten days. The perforation of cornea took place in the day after the case was first seen, and the globe became subsequently quite shrunken. No history of direct contagion could be obtained in this case, nor did it appear that the disease was propagated by the patient to any other persons.

The second case was apparently much milder as far as the amount of swelling of lids was concerned, but the appearance of the membrane on the palpebral mucous surface was quite characteristic and there was a good deal of chemosis. The patient, a little girl about eight years old, was treated as an in-patient, and at the time of admission the cornea was entire and bright. The quinine lotion was diligently used, but the disease appeared to increase steadily, and the cornea ulcerated and perforated, and it was not until about the twelfth day that the

false membrane ceased to form, leaving the lids very granular and they remained so for several weeks.

A sister of this patient developed a mild attack of the disease which yielded speedily to treatment.

To those who have seen this disease in towns where it is frequent, and it is alleged to have occurred in this country, in Manchester, the above cases may appear uninteresting and commonplace, but they are the only examples of this disease that have come under my observation in thirteen years' hospital out-patient practice, and I have direct assurance from ophthalmic surgeons to other hospitals of similar experience.

It is to be regretted that fuller details of the constitutional condition of these patients is absent, but the following questions suggest themselves. Do patients ever manifest any paralytic phenomena either of eye, throat, or limbs after diphtheritic ophthalmia? And is the presence of albumen in the urine frequent either during or after an attack?

I should mention that I, in common with the rest of the profession, am entirely indebted to Mr. John Tweedy for the suggestion of the quinine lotion for these cases, and the recording of the above cases is not intended to imply that his treatment is not the best at present known. I was not aware until after writing the above that his papers in *The Lancet* for January 24, and February 21, 1880,* contained a description of a case as typical as those recorded here successfully treated after his plan; and if another case occurred to me I would follow again this course, perhaps more energetically.

* "On the treatment of Diphtheritic Conjunctivitis by the local application of Solution of Sulphate of Quinine, by John Tweedy, F.R.C.S."—*The Lancet*, 1880, vol. i, pp. 125 and 282.

## ON SOME UNUSUAL FORMS OF IRITIS.

By J. E. Adams, F.R.C.S.

About three years ago I mentioned in a communication to the Hunterian Society three cases of iritis occurring in young girls about the age of puberty in whom I was utterly unable to find any dyscrasia to account for the occurrence. Two out of the three happened in private practice, and I became acquainted with the personal history of the parents and brothers and sisters in great detail, and no history of gout, rheumatism, or syphilis could be reached. All three patients were types of childish health and beauty, and the characters of the disease were identical in all. It is characterised by an abundant effusion of lymph producing firm posterior synechiæ, a total absence of pain or photophobia, and great injection not limited to, or visibly intensified at, the ciliary region, and involving the conjunctiva so that one and all had passed for some time while under treatment by the medical attendant as cases of conjunctivitis. The disease subsides apparently of its own accord, leaving the eye scarcely at all impaired; two having V = $\frac{20}{20}$ and Jaeg. 1, and one $\frac{20}{40}$. They were all unilateral and on the left side. Recently another precisely similar case has occurred. In this case there are decayed teeth in the upper jaw on the same side, but in the other the teeth were good.

Another very remarkable case of iritis occurred in a patient, a gentleman twenty-nine years of age, free from gout, rheumatism, or syphilis, but living in a marshy country and subject to intermittent fever. At the time of observation his left eye, for which he consulted me, showed marked ciliary redness, though the pupil was active, and the media clear. V = $\frac{20}{20}$. Spots of uveal pigment on right capsule.

In a few days a few posterior synechiæ were revealed in the left under atropine. Ordered atropine and iodide of potassium. After the lapse of a week he was worse, complaining

of considerable pain, which he said was always worse at two o'clock in the morning.

This periodicity was so marked that the conviction that some miasmatic influence was at work was forced upon me, and I prescribed quinine, with the result that immediate relief was obtained, and the eye assumed its normal appearance in a week. Since then (now four years) he has had one or two slight relapses, but timely doses of quinine have always brought the complaint to a speedy ending.

## HOUSE SURGEON'S NOTES.—TREATMENT OF IN-PATIENTS.

By G. Herbert Burnham, M.B.,

*Senior House-Surgeon.*

### *Use of Carbolic Acid Lotion in Gonorrhœal Ophthalmia.*

Being desirous of testing the curative power of the above mentioned lotion in this variety of ophthalmia, I was allowed to do so in the following cases, through the kind permission of the surgeons.

Elizabeth H—— æt. 21, admitted under the care of Mr. Lawson, became an in-patient in June, 1880. On admission the condition of the left eye was as follows:—The lids were much swollen, brawny, and so tense that the upper lid was everted with much difficulty, and then not fully; the ocular conjunctiva chemosed and overlapping so extensively the cornea that its margins could not be seen, though several attempts were made. There was also a copious purulent discharge. The cornea as far as visible, was unimpaired. The right eye was also affected, but not nearly so severely, and never equalled the other in intensity. The patient had light, flaxen hair, a thin, pale skin, and was decidedly anæmic looking, in fact, presenting the appearance of a person of by no means vigorous habit of body.

The treatment pursued was the frequent and free use of carbolic acid lotion, one part of the acid in twenty parts of water, and cleansing with iced water. The mode of application was as follows:—On admission, the lids were everted and thoroughly bathed with the lotion; the girl was also ordered to keep the eyes clean by means of the iced water, and to apply every half-hour, at least, the lotion to the eyes. In addition, I applied the lotion to the everted lids twice daily, and the nurse four times during the twenty-four hours. In twelve hours improvement was visible. As at the end of forty-eight hours the improvement still continued, and the eye-lids smarted, I ordered that she herself should use the carbolic acid solution (one in forty), the stronger solution (one in twenty) being still continued by myself and the nurse. The constant use of the one in twenty solution was soon returned to, as the conjunctival chemosis

was again increasing, as also the discharge. The free application of the above treatment was kept up for one week longer, being comfortably borne, when she was allowed to use the weaker solution a portion of the time. The girl was discharged one month from admission almost completely cured, that is, the discharge was almost nil, and the appearance of the eyes normal, save a little injection. When seen on subsequent occasions, the cure seemed permanent.

*Note.*—The carbolic acid lotion must be thoroughly applied, at least six times in the twenty-four hours, to the everted lids, bathing them with it as freely and unsparingly as with water; and the patient must also be taught to hold the lids well apart when using the lotion, and also to keep up the application during the night time. The use of the lotion is followed by smarting, not at all severe, and a feeling of warmth in the eye, both of which disappear in a few minutes. The cornea in this case continued throughout healthy. When the disease is still severe, the constant use of the one in twenty solution is necessary, as witnessed in the increased conjunctival chemosis under the use of a weaker solution.

A second case, a young man, was admitted a short time ago under the care of Mr. Wordsworth, in which the same treatment was followed throughout. In this case, though the swelling of the lids was not as great, that of the ocular conjunctiva was as marked, and also more firm and fleshy. On his admission there was in one eye a deep and large excavation of the cornea with slight infiltration of the margins, and falling forward of the iris against its posterior surface. The excavation began very quickly to improve. The improvement was as decided and almost as rapid as in the first case. The lotion (one in twenty) was freely used throughout, that is, a pint or so was poured into a dish, and then the everted lids were freely bathed with it by means of absorbent wool. In addition, at least every half-hour during the day-time, and many times during the night, the patient used it thoroughly, in so doing keeping the lids well apart and the head tipped a little backwards, so as to allow free ingress.

The third and last case was that of a young man, also under the care of Mr. Wordsworth. On admission, the swollen lids on being everted exposed the palpebral conjunctiva, much thickened and spongy looking; the ocular conjunctiva was very much

swollen, lying in great folds, overlapping the cornea so much that its central third only could be seen, and the edge of an excavation could be just perceived. The discharge was very abundant. The right eye, being unaffected, was covered with Buller's eye-shield.*

As in the other cases, improvement was soon noticed; and in twenty-four hours the conjunctival swelling had so gone down, that the greater portion of the cornea, with a deep excavation at its upper part, could be seen, only, however, when the eyeball was moved in different directions by the patient, as, when at rest, the conjunctiva overlapped. At the end of seventy-two hours the following note was made: The whole cornea is now visible; there is seen a deep excavation of the upper third, the edges of which are just beginning to lose their sharply defined outline. The rest of the cornea is normal looking. A day or so later small vessels were making their appearance on the excavation; and the whole eye looked so well, that all danger of the giving away of the cornea was thought to be passed. At the end of eight days the progress was still as satisfactory in every way. The treatment was the same in every particular as in the case of the other young man.

*Remarks.*—In regard to the first case, subsequent experience seemed to show that improvement would have been more rapid if the one in twenty solution had been used throughout.

In the second case, the condition of the patient at his discharge, eighteen days after admission, was as follows:—The lower iris still in contact with the cornea; no tendency of the corneal excavation to bulge, in fact, daily looking less likely to do so; the upper part of the chamber normal in depth, and the corresponding cornea perfectly clear; the lids were but slightly gummed together after several hours sleep.

The condition of the third patient, fourteen days after admission, when he was discharged, was as follows:—No swelling of the lids: their conjunctival surfaces quite normal in appearance; the ocular conjunctiva still injected, and

* The Lancet, May 16, 1874, and in Mr. Lawson's Manual, 4th Ed., p. 10.

slightly thickened towards the outer canthus. The cornea was quite clear below; at the upper part was the healing excavation, freely supplied with numerous small vessels running along its floor. The anterior chamber was normal in depth. After sleeping several hours, the lids were but slightly stuck together.

In carbolic acid lotion, in the form of a *strong solution*, we seem to possess a powerful and convenient remedy for Gonorrhœal Ophthalmia. It gives the patient but little discomfort, and exercises a marked astringent as well as antiseptic influence, together with a beneficial action on the cornea, especially in cases in which there are more or less advanced semi-transparent excavations, so often ending in perforation, or even destruction of the cornea, under other modes of treatment.

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## No. II.

### *Corneal Infiltration with Hypopyon.*

Three modes of treatment were applied to these cases. Some cases were treated with eserine (gr. ij ad. ʒ i.) instilled four to six times daily, and by frequent bathing with decoction of poppies. If there was much pain, leeches (generally two at a time) were applied to the temple of the affected side. Internally, sulphate of quinine was administered in two-grain doses three times a-day.

In other cases, there was added an operative procedure to the above treatment. This consisted in making an incision with a Graefe's knife along the entire width of the base of the ulcer and through the thickness of the cornea. In those cases in which the puro-lymph did not come away from the anterior chamber with the gush of aqueous, it was coaxed out, or if too thick to flow, was pulled through the opening by small forceps. The removal of the pus from the anterior chamber, especially if it were thick, appears to have increased the beneficial effect.

The last form of treatment to be mentioned was the

instillation of atropine drops three times daily, frequent bathing with belladonna fomentation and quinine internally.

In corneal infiltration with hypopyon, the corneal incision through the base of the ulcer, with subsequent eserine treatment, seems to give the most satisfactory results. Where the corneal infiltration is small and the hypopyon slight, the eserine, or first mode of treatment, was alone used, as improvement took place rapidly; but in cases in which the extent of each was considerable in amount, or seemed actively increasing, the corneal incision in addition certainly gave the impression of accelerating the rapidity of healing.

In comparing the cases treated with eserine, with those treated with eserine and corneal incision, and selecting from each those which closely resembled one another, operative interference seemed not only to hasten a favourable result, but also to lessen the size and denseness of the corneal cicatrix.

With atropine, the progress towards a favourable termination was slower, the tardiness of the healing process exhibited being in marked contrast to the activity shown under eserine.

The rapidity of the beneficial effects of eserine was very marked in those cases of infiltration of the cornea with but little, if any, conjunctival injection. It would seem that in these apathetic conditions it proves itself superior to atropine, not only by its quicker healing power, but also by giving a more sharply defined boundary to the infiltration. Transparent excavations of the cornea were also most satisfactorily influenced by it, the sharply defined edge soon becoming rounded. In some cases the corneal incision has been repeated in different places with advantage. The necessity of the repetition arises by reason of the infiltrations being isolated, and hence there is an inability to deal with more than one at a time. It has been observed that in such cases the infiltration so tapped has more rapidly healed than the other not so treated; and that the incision of the second was followed by equally good result. Sometimes the opening of the incision once a day for a few times is beneficial, especially in cases in which a reforming of the anterior chamber is

accompanied by hypopyon, coming to all appearances from the same place as at first, that is, where the incision was first made.

The danger of an anterior synechia is very slight, if after the performance of the incision a firm compress bandage be applied to the eye for about ten hours, as by this time, if the bandage be left undisturbed, the anterior chamber has reformed. In some few cases in which the incision was opposite the margin of the pupil, the instillation of atropine once or twice dilated the pupil sufficiently to drag it away from the opening; as soon, however, as the chamber is of some depth, eserine is to be resumed. It may be that there is also less tendency of the iris to adhere to a wound through tissue so infiltrated.

May not the action of eserine in these cases be partly explained by its powerful effect on the accommodation of the eye, calling it into greatly increased activity, and thus bringing about an unusually large vascular supply to parts in close vicinity to the cornea, in this way giving to the cornea a greater supply of blood from which to draw sustenance? The often repeated instillation, by keeping up continually the spasmodic condition, retains without intermission the heightened vascular supply. From this continuance there results a brighter corneal lustre, and also at times the presence on the cornea of vessels so minute that they can only be seen by oblique illumination and using a second lens as a magnifier.

In cases in which there are much photophobia and conjunctival injection associated with the corneal mischief, atropine often acts better than eserine, as here it gives the needed rest by paralysing the accommodation.

If iritis be present, eserine is generally regarded as contra-indicated, the beneficial effect of the eserine not being then as marked as without the iritis, while it gives a small pupil more easily bound down by adhesions. Atropine does away with this danger to a great extent, and seems to have as great curative action on the corneal affection as the eserine, and

in some cases greater. In a few cases, in which iritis did not seem to be present at first, unsatisfactory after-progress with eserine was found to be associated with iritis, as the pupil did not under atropine assume either its full or normally round dilatation. Under atropine, however, improvement began and steadily progressed. In a few cases of corneal infiltration with some conjunctivitis either with or without iritis, the solution of carbolic acid (one in twenty) has been used with such good effect, that further trials are being continued.

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## No. III.

*Entropion following Granular Lids; Treatment by Bürow's Operation.*

THE entropion arising from the cicatrices of old granular lids has been treated with good results by Bürow's operation. This, as practised at Moorfields, consists in making an incision along the inner surface of the lid, through the entire thickness of the cartilage of the upper lid, into the muscular and areolar tissue, following as a guide the cicatricial line which is usually seen running along the conjunctival surface, near to the free border of the lid, from almost the outer to the inner canthus.

The operation is begun by everting the upper lid on a small spatula, resembling a diminutive shoe-horn, which is kept behind the lid when so everted. The lid being held in position on the horn by the index finger, an incision is made in the cicatricial line, and extending, when completed, from well up to the outer canthus to close into the inner, and through the entire thickness of the cartilage, leaving intact, however, the integument of the lid. The horn being pressed firmly down into the groove caused by the everted lid, and the latter pulled as far up as possible on it, not only lessens the bleeding, but also causes the incision to gape, and thus enables one to see when the cartilage is

almost divided, giving in this way an indication for cutting cautiously so as to avoid injuring the integument.

Another way of doing the operation is to make a small incision through the cartilage of the everted lid from the conjunctival surface about the centre, and then, passing one blade of a pair of scissors through the hole, to slip the blade along between the skin and cartilage, and then on closing the blades the cartilage included between them will be divided. This division should follow the same direction as that mentioned above, and be as complete.

In this operation it seems necessary, for a perfect result, to leave no bridge of cartilage undivided, to extend the incision well up to each canthus, to have the cicatricial line well-marked, and to follow it accurately in making the incision. When the section has been made, the edge of the lid with the lashes should lie in a normal position, and a blue line caused by the extravasted blood should be seen through the integument corresponding to the incision in the cartilage.

This operation has been often done, and in a number of cases, extending over several years, has been attended with most satisfactory results. In some of the earlier cases the after-effect was slight; but experience seemed to prove this to have been due to want of thoroughness in the performance of the operation. It is perhaps not advisable to perform the operation till the cicatricial line is well marked, as failure in some of the early cases may have been due to making the incision in a wrong position, owing to the absence of the guide afforded by the cicatrix.

As the keeping of the lid in the proper position on the horn with the index finger, is not only tiresome, but also sometimes difficult, owing to the contracted condition of the cartilage, I have devised an instrument (see fig.) to alleviate the inconvenience. It consists of a horn, similar to the one used with the finger, and a movable metallic bar fastened to the horn, and so contrived as to take the place of the finger, both in freedom of move-

ment and power of holding the lid in position. Towards one end of the horn a slit is made, and through this there passes

a screw, not only securing one end of the bar, but also enabling it to be fastened in any part of the slit, so as to regulate the position of the other end. This screw is usually left loose, so as to give freedom of movement to the other extremity in any desired direction. The free end, which is to grasp the upturned lid, is toothed and also bevelled, so as to allow a better view of the exact position of the teeth, and hence admitting the cartilage to be grasped close to the free border, and at the same time firmly. This is practically of importance, because the surface between the cicatrix and the free edge is sometimes very narrow, and if the cicatricial line is to be closely followed as a guide, it ought not to be encroached upon. The widened part of the bar is for the tip of the finger to rest upon, and is roughened to afford greater security of grasp. A spring is placed beneath, so as to allow of quick and easy elevation of the toothed extremity.

The central part of the cartilage corresponding to the width of the horn is first completely divided; and then, by shifting it to one end of the lid and then to the other, the division of the whole cartilage is easily completed. The lateral movement of the serrated end is now of use, as a firm hold can be taken of the cartilage at each end of the lid. The knife used to make the incision with is a Sichel's or a Beer's. The instrument has been made for me by Weiss and Son.

## CURATOR'S PATHOLOGICAL REPORT.

By W. A. Brailey, M.D.

*Tumours of the Eyeball or Orbit:* 28 *cases.*

These are classified into **A.** Tumours not encroaching upon the Cavity of the Globe. **B.** Intraocular Tumours. These last comprise (*a*) 13 cases of Sarcoma of the Uveal Tract and (*b*) 10 cases of Retinal Glioma.

A.—*Tumours not encroaching upon the Cavity of the Globe:* 5 *cases.*

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| No. 548. Mary G., æt. 62. Under care of Mr. Lyell. A rounded unpigmented tumour grows from the inner sclero-corneal junction of her *Right* eye. It is the size of a large bean and overlaps the cornea by its anterior thin edge, as well as the sclerotic by its posterior thicker part. The globe looks otherwise | Tension normal. Posterior arterial circle in ciliary body is slightly enlarged. The anterior is about normal. | A small round-celled sarcoma is found in the deeper layers of the sub-conjunctival tissue, being in contact with the proper scleral and corneal tissues on the one hand, but separated on the other from the conjunctival epithelium by the unaffected superficial loose connective tissue web. It extends back for some distance as a thin layer in the episcleral tissue, besides overlapping the sclerotic behind and the cornea in front. The unchanged epithelium of the conjunc- | The ciliary folds are not shrunken. The most anterior of them show some some slight nuclear increase. The ciliary muscle is of good size and shape, and its fibres are slightly inflamed. There is a general increase of nuclei in the episcleral tissue, especially near the tumour, and a slight similar change in the tissue of the sclerotic and cornea. The papilla is slightly swollen, there is some excess of nuclei in it, which appears to be in the walls of fine blood-vessels. | .. .. | The changes in the O. N. are more conspicuous than those in the ciliary region. Six months after the excision of the globe, there was no recurrence. Six months later she returned with the orbit filled with a firm mass of tumour. |

A.—*Tumours not encroaching upon the Cavity of the Globe*—continued.

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| No. 548.—*continued.* | | | | | |
| healthy as far as concerns its exterior. The pupil is round and mobile. Near vision = J. 6. The eye waters very much. The tumour has been removed twice; The last time a year ago. | | tiva can be traced continuously over it. | There is an increase of nuclei in the connective tissue of the anterior part of the nerve and increased connective tissue in the nerve further back, most developed in relation with the blood-vessels. There is no evidence of any inflammation in the retina, nor of any proceeding back along the choroid from the ciliary region to the papilla. | | |
| No. 388. Edward T., æt. 60, under care of Mr. Streatfeild. His *Left* eye presents a darkly pigmented sarcoma the size of a small bean, growing on its external surface at the sclero-cornical junction of the outer side. Its | Tension normal. Arteries of ciliary body slightly enlarged in the neighbourhood of the tumour. Elsewhere they are normal. | The conjunctival epithelium can be traced completely over the tumour which is constituted by an infiltration of the sub-conjunctival tissue by cells which are ·1 mm. wide and drawn out at each end into a short tail. Their broadest part is constituted by a large staining nucleus with a narrow external zone of | The ciliary body and iris, where they are nearest to the tumour, are a little inflamed, the muscular fibres more especially. The ligamentum pectinatum is slightly inflamed but not contracted. The ciliary folds are of good size. The periphery of the anterior chamber of the aqueous is of fair depth. | .. .. | .. .. |

A.—*Tumours not encroaching upon the Cavity of the Globe*—continued.

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| No. 388.—*continued.* surface is marked into rounded lobules. It was first noticed as a red spot 10 years ago. | | protoplasm. The adjacent episcleral tissue contains numerous round nuclei which are only half as large. These become more rare as we pass into the sclerotic, from the corpuscles of which they become indistinguishable. No tumour cells are found anywhere else. | The canal of Schlemm is large. The ciliary body and iris, where they are farthest removed from the tumour, are in a similar condition, though their arteries are scarcely at all enlarged. The O. D. is not swollen. There is slight but distinct inflammation of the connective tissue in the nerve trunk. The episcleral tissue has its nuclei somewhat increased. | | |
| No. 56. Joseph B., æt. 60, under care of Mr. Hulke. There is an epithelial growth at the outer sclero-corneal region, which was first noticed 4 years ago. Several ineffectual attempts have been made | Tension probably normal, but globe extremely tender. The ciliary arteries are a little enlarged in the ciliary body near the tumour. | The conjunctival epithelium is thickened, gland-like extensions from it run down into the loose sub-conjunctival connective tissues like solid columns of cells running in various directions, which are cut in the sections at all sorts of angles. The episcleral tissue is loosely infiltrated with small round cells, | There is slight but distinct inflammation of the connective tissue of the O. N., and of the fibres between its inner and outer sheaths. The ciliary body is inflamed, especially its muscular fibres, as is also the iris. There is a little atrophy of both. The ciliary folds are small and not advanced. The peri- | The sclerotic presents a slight nuclear increase, especially in its outer layers and in the walls of its blood-vessels. This is especially seen in the | .. .. | |

A.—*Tumours not encroaching upon the Cavity of the Globe*—continued.

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| No. 56.—*continued.* to remove the growth previous to the excision of the eye. | | some of which are multinuclear. | phery of the anterior chamber of the aqueous is shallower than normal, but the cause of this is not apparent with certainty, though the ligamentum pectinatum appears a little inflamed and somewhat contracted. | sub-conjunctival tissue. | |
| No. 577. Edward L., æt. 18, under care of Mr. Adams. Proptosis of *Right* has been noticed 5 months. *At excision*, the vision is perfect (= $\frac{6}{6}$ and J. 1). The O. D. looks normal. | Tension normal. The arteries of the ciliary body are a little enlarged. | There is a tumour behind the eye of doubtful origin. Its connections appear to be with the periosteum. It had to be removed in fragments which were firm and yellowish-white in colour. The tumour is made of a firm, fibrillated tissue containing many nuclei, mostly elongated. There are, however, small areas in the midst of the fibrous tissue where the nuclei are all round and so abundant as to nearly replace altogether the fibrous tissue and give | The O. N. presents a slight increase in the fibrous tissue of its trabeculæ and in their nuclei and blood-vessels. The papilla is a little swollen at its margins. The ciliary body and iris are very slightly inflamed, especially their muscular fibres, but not atrophied. Indeed the connective tissue internal to the muscle is scarcely at all affected. The ciliary processes are enlarged and advanced a little so that the peripheral part of the | .. .. | The patient died from a recurrence in the orbit 2 months later. |

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| No. 577.—*continued.* | | the appearance of a small round-celled sarcoma. The blood-vessels are not very numerous. | anterior chamber of the aqueous is rather shallow. This might be due to the pressure of the ciliary folds on the iris. The canal of Schlemm is of good size, and the ligamentum pectinatum very little if at all contracted. | | |
| No. 570. Samuel C., æt., 65, under care of Mr. Lawson. The *Left* eye has been pushed forwards for 4 weeks only. The cornea is clear, the anterior chamber of good depth, the pupil dilated and inactive. There is no *p.l.* The ophthalmoscope shows decided swelling of the papilla. | Tension very slightly increased at excision. He says it has been harder at an earlier period. Arteries of ciliary body enlarged, especially its posterior circle. The circulus major is surrounded by | A sarcoma, made for the most part of small round cells deeply staining throughout, is found attached to the outer nerve sheath, though it is not clear that its only connections are with this. It gradually blends with its fibrous tissue. | There is decided hypertrophy of and connective tissue increase in the fibrous frame-work of the O. N. Its nuclei are also increased in number. The ciliary body is considerably inflamed, and its muscular bundles are very atrophic. The iris is slightly inflamed and somewhat atrophied. The peripheral part of the anterior chamber of the aqueous is obliterated by the application of the peripheral part of the iris to the outer- | The tumour recurred in the orbit shortly after excision of the eye. Tumour cells were found in the glands of the neck and in the liver. The pia mater all over the brain was minutely injected. There | His urine was not examined, but there was no reason to suspect renal disease. |

A.—*Tumours not encroaching upon the Cavity of the Globe*—continued.

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| No. 570.—*continued.* | an excess of nuclei. | | most or most posterior part of the ligamentum pectinatum. The ciliary folds are much distended with enlarged capillaries. They are probably larger and more advanced than normal. The episcleral tissue is inflamed, especially near the sclero-corneal junction. There is no disturbance of the pars ciliaris retinæ or of the vitreous body. The retinal arteries have their walls enormously thickened with the "hyaline fibroid" change which affects its entire coat, except the innermost layers. There is a slight nuclear increase in the choroid generally. | was lymph effused over the anterior two-thirds of the upper surface of the brain. The increase of nuclei in the episcleral tissue extends all the way from the O. D. to the ciliary region. It is greatest in front. There is also a slight increase in the sclerotic and cornea generally. | |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of Uveal Tract.* 13 *cases.*

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| No. 580. James L., æt. 52; under care of Mr. Lawson. Dimness was noticed in his *Left* eye 17 months ago. 13 months ago his retina was detached at its upper and inner part; the pupil was inactive; there was occasional pain. He read J. 19 only at 12″. *At excision*, the cornea was clear, the pupil large and round. There was no *p.l.* | Tension slightly diminished but not to minus 1. The circulus arteriosus iridis major is a little above average size. | The tumour fills half the cavity of the globe projecting as a rounded mass from the inner part of the choroid. It overhangs the O. D. behind, pressing together the 2 folds of the retina, and in front extends beyond the equator. The cells of the tumour are spindle-shaped, and rounded lumps of brown pigment are scattered among them. The few blood-vessels that are met with have very thin walls which do not appear to be surrounded by a sheath space. The tumour is probably derived from the middle and outer layers of the choroid, for the chorio-capillaris can be traced for some distance unchanged over its internal surface. | The ciliary body and iris are slightly inflamed, but not at all atrophied. The ciliary folds are large and somewhat advanced, so as to be very near to the iris which in its turn is very near to the cornea, thus rendering shallow the peripheral part of the anterior chamber of the aqueous. The ligamentum pectinatum is a little inflamed. There is slight optic neuritis, *i.e.*, the connective tissue framework of the nerve is somewhat inflamed. The retina at the papilla is pushed aside, and atrophied on account of the presence of the tumour. | There is an excess of small staining nuclear bodies in the part of the sclerotic adjacent to the tumour, which condition extends to a less degree all the way to the O. D. | 3 months later there is no recurrence. The Right eye, however, is weak and irritable. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of Uveal Tract*—continued.

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| No. 293. Emma T., æt. 42; under care of Mr. Adams. The vision of her *Right* eye has been noticed to be defective for 3 years. *At excision*, the cornea was clear; the ant. chamber of fair depth; the iris a little thickened and discoloured. The pupil dilated circularly with atropine. There was doubtful *p.l.* | Tension as above. Arteries normal. | A rounded firm tumour, the size of a hazel nut, with a slightly lobulated surface projects from the choroid of the posterior part of the globe, and overhangs the O. D. Its central part is mottled with pigment. The retina generally lies *in situ*. It is adherent closely to the summit of the tumour. | The ciliary body and iris are a little inflamed, but of good size and shape. The ciliary folds are large, and may be a little advanced, though clearly removed from the iris, whose peripheral part is a little nearer the corneal margin than usual. The canal of Schlemm is very well seen. The ligamentum pectinatum may be a little contracted. There is slight inflammation of the nerve trunk as above. The tumour has the same structure and relation to choroid as in the case above (No. 580). It is, however, much more vascular than that, and contains very little pigment. | .. .. | No answer was returned by the patient or her friends to a subsequent enquiry. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of Uveal Tract*—continued.

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| No. 313. Lucy S., æt. 13, under care of Mr. Adams. The *Left* eye was noticed to have something wrong with it 3 months ago. *At excision*, there were 2 yellowish nodules on the anterior surface of the lower part of the iris, very like to syphilitic gummata in appearance. There were point-like posterior synechiæ especially at the lower pupillary margin. | Tension slightly diminished as above. Arteries normal. | The lower limb of the iris is vastly thickened, except at its apex, into a tumour whose anterior border is close to the posterior corneal surface. The tumour is made of small round cells which are supported by a fine imperfectly fibrillated reticulum, with very rare blood-vessels. At points, the cells are more closely clustered, and less deeply stained, forming denser rounded nodules in the tumour mass. Similar rounded lumps are scattered through the otherwise uninvolved ciliary muscle. Some of the ciliary folds are distended with similar cells. A few similar rounded masses of cells are found in the ciliary body, even where it is farthest removed from the tumour. | The iris and ciliary body are a little inflamed, but not at all atrophied. Indeed at the lower part of the ciliary circle, the ciliary body is thickened by the swelling of it, and the ciliary folds from the presence of tumour cells, so as to be nearly double its usual diameter. The iris is thickened many-fold. The optic disc is a little swollen, and contains many fine blood-vessels. There is very slight excess of nuclei in the nerve-trunk. The lens is *in situ*. The space between the lens and ciliary body below, as also the lower part of the anterior chamber of the aqueous, are nearly, if not quite, obliterated. | The tension is even below the normal, notwithstanding the obstruction at the circumlental space, and at the entrance to Schlemm's canal. | The patient is healthy-looking. There is no family history which can throw any light upon the case. Sometime afterwards, however, her friends report that she has become the subject of pulmonary phthisis. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of Uveal Tract*—continued.

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| No. 375. Annie C., æt. 19, under care of Mr. Adams. The outer part of the field of vision has been noticed to be defective for 9 months. *At excision* V = $\frac{20}{20}$ barely, and Jaeger 1. The pupil dilates round with atropine. The anterior ciliary arteries, as seen on the exterior of the globe, are much distended. | Tension normal. Circulus arteriosus major perhaps a little above average size in places. | A large rounded darkly pigmented tumour projects from the inner surface of the ciliary body into the cavity of the vitreous. It is made principally of dark brown spherical pigment masses of enormous size (·022 mm., after the hardening of the preparation in Müller's fluid and alcohol), which are in some places aggregated by themselves loosely into groups, whereas in others they have intermixed with them, some smaller cells (·01 mm.) with distinct small nuclei. There are also sometimes small lighter coloured pigment cells, and a few nuclei which appear to be free. There is no stroma other than that formed by the few blood-vessels, and in places the remains of the structure of the ciliary muscle. The capillaries are large and thin-walled. | The ciliary body at the inner part of its circuit is enormously thickened at its posterior part by the tumour, its anterior part is tilted forwards, so that the iris periphery is very near to the cornea. Still the canal of Schlemm is visible, and access to it is possible, except at one point where the meshes of the ligamentum pectinatum are invaded by pigment cells. The iris is inflamed. Further from the tumour the iris and ciliary body are somewhat inflamed. There is no atrophy of either. The ciliary folds are decidedly large and near to the iris, which is nearer to the cornea than normal. The optic papilla is somewhat swollen, and contains very many fine blood-vessels. There is distinct, though slight, neuritis of the nerve trunk. | The sclerotic near the tumour contains a decided increase of nuclei which extends all the way, especially in the walls of its blood-vessels, to the optic disk. | There was no recurrence. At the extreme margin of the tumour there is seen simply an increase in the size and number of the pigment epithelium cells on the internal surface of the ciliary body. Nearer its centre, large round pigment cells appear in the space between the ciliary body and sclerotic. Nearer to the tumour - centre the two sets of pigment cells are continuous, and |

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| No. 375.—*continued.* | | The arteries have no sheath-space surrounding them. | | | they extend forward for a considerable distance between the fibres of the ciliary muscle destroying its structure. |
| No. 612. M. B., æt. 84. *Right* eye excised and presented by Mr. Bowman. 13 weeks ago there were incipient cataracts in both eyes. The *Right* had T. n.; and very dull fundus reflex, the outer part of the field was wanting. J. 8 was read badly at 6″ with $+\frac{1}{60}$. *Four weeks* later the eye had | T. very much increased (T. 3). Arteries forming the posterior ciliary circle much enlarged. Little if any enlargement of the anterior circle. | The retina lies detached in loose folds. A sarcoma projects from the inner equator into the cavity of the globe, as a thickening of the choroid. Its most anterior part, which is steep with rounded summit, is 7 mm. high, and overhangs its base somewhat, but the ciliary region is not nearly reached by it. Behind it shades off gradually into the normal choroid on the outer side of the O. D. The tumour is made of cells, | There is great atrophy of the ciliary body, especially of its muscular fibres, as well as a distinct nuclear increase. The iris is much inflamed, though less atrophied. Its peripheral part is adherent to the cornea. It is greatly advanced in front of the rest of the iris, and is strongly curved forwards, as if by some pressure from behind. The ciliary folds where furthest removed from the tumour are not shrunken. Nearer the tumour they are some- | .. .. | .. .. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of Uveal Tract*—continued.

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| No. 612.—*continued.*<br><br>still T.n, but doubtful p.l. 5 days before excision, the patient came with pain of 3 weeks' duration. Scleral injection and T. 3.*<br><br>* The microscopical specimens of this case were prepared by Dr. L. W. Fox. | | | whose large round or oval nucleus is surrounded by a protoplasmic zone of very irregular outline. There is no stroma between the cells, though rare bands of fibrous tissue, apparently the remains of the connective framework of the choroid, traverse the tumour. Some blood-vessels are met with, and they are apparently surrounded by a sheath-space. In front of the anterior abrupt termination of the choroid, there are scattered a few rounded masses of cells of similar character, as well as some smaller cells still more in front, arranged in lines between the central choroidal lamellæ. The choroid on either side of the O. D. is invaded, and nearly destroyed by similar cells | what atrophied. In neither case are they advanced, but there is always a wide interval between them and the peripheral part of the iris. The O. N. contains a great increase of connective tissue, especially round its blood-vessels. There are large nuclei in it, principally arranged in rows, which may occupy the place of the remains of the nerve-bundles. They have some similarity to, and might be taken for, tumour cells. | |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 612.—*continued.* | | arranged in similar rounded groups. Smaller editions of the same are seen in the inner retinal layers. | | | | |
| No. 470. Henry D., æt. 37, sailor. Under care of Mr. Streatfeild. He found out accidentally 6 months ago that the outer half of his field of vision was deficient. He had no other inconvenience till a month ago, when he had pain in the eye, lasting more or less till its removal. *At excision*, conjunctiva inflamed and chemosed: pupil widely dilated. The retina is detached; the lens and choroid are *in situ*. | Tension increased. Arteries of ciliary body dilated and crowded with red blood corpuscles. | A tumour projects from the choroid of the inner side, involving the inner part of the ciliary body, where it is 10 mm. thick. Its posterior end, which shelves off gradually, is 5 mm. removed from the O. D. The tumour, where it is thickest, has a narrow internal zone like an unaltered chorio-capillaris. In its centre it consists of smallish, round, deeply staining unpigmented bodies, mixed with larger pigment masses, some of which are elongated, though most are rounded. In its external layers these last predominate, and some of them are found in the inner part of the adjacent sclera. The tumour has | The O. N. has its fibrous trabeculæ hypertrophied, and its nuclei are increased in number and in size. The same in the inner and outer sheaths of the O. N. and in the loose tissue between them. The side of the O. N. farthest removed from the tumour contains a still larger number of nuclear bodies, not in its fibrous tissue alone, but also within its nerve-bundles. The retina contains a slight excess of round nuclei in its inner layers. There are no pigmented cells either in the retina or in the O. N. The ciliary body is much shrunken and fibrous in structure. The ciliary folds, though much shrunken in | .. .. .. .. | |

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| No. 470.—*continued.* | | a scanty fibrillated stroma and many large thin-walled blood-vessels. At its posterior thin edge the smallish round unpigmented cells are seen to extend into the outer layers of the surrounding choroid and the adjacent scleral layers. The middle choroidal layers contain a slight increase of their pigment. There is also a general thickening of the choroid due apparently to a thickening of its fibrous lamellæ. The choroid nearer the O. D. presents no increase in its thickness, and no excess of nuclei. The sclerotic presents a general nuclear increase, especially in the loose tissue on its exterior, where the nuclei are very like the smaller cells of the tumour. | volume, are advanced so as in places to be close to the peripheral part of the iris, to which, however, they are not adherent. The iris is atrophied, and its peripheral part is applied to the cornea. | | |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 121. Lucina G., æt. 29, under care of Mr. Hulke. For 2 years her *Left* eye has been getting gradually dim. Six days ago it became painful. The pupil was semi-dilated and fixed. This case is mentioned in its relations to increased tension in vol. ix., p. 225. | Tension increased. Arteries of ciliary body enlarged. | The retina is detached, but not contracted. On the outer side is a choroidal sarcoma, extending from 5 mm. behind the ora serrata to within 2 mm. of the O. D. Its most internal part, the size of a small bean, is connected by a narrow neck with a broader base, which shelves gradually into the surrounding choroid. Microscopically the tumour is a sarcoma composed of small round cells. | The ciliary body, especially its muscular fibres, and the iris are inflamed. The first is a little atrophied. The ciliary folds, however, are not shrunken; they are adherent to the lens-capsule, and sometimes appear to be somewhat advanced so as to be near to, at times in contact with, and even adherent to the posterior surface of the iris, whose extreme peripheral part is adherent to the extreme corneal margin. The posterior pigment epithelial layer near the more internal part of the iris is adherent to the front of the lens-capsule. There are no microscopical specimens of the O. D. | .. .. | .. .. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 26A. Female, æt. 37. Specimen received from Dr. Bell Taylor. Three years ago the sight of the eye failed and a tumour was diagnosed. | Tension and arteries as above. | A sarcoma of the outer part of the choroid extends from the ciliary body to the O. D. It appears to be constituted by a thickening of the middle and outer choroidal layers by small oval unpigmented cells, and also by larger bodies containing pigment. The latter are found especially in the outer layers of the tumour. It contains also a moderate number of thin-walled blood-vessels. The chorio-capillaris does not appear to be much affected, but can be traced in sections as a thin zone on the inner surface of the tumour. There are places in the tumour where the unpigmented cells appear to be degenerated, and thus unstained granular areas are formed, surrounded by a zone in which pigment granules are abundantly scattered. | The ciliary body and iris are inflamed and somewhat atrophied. The iris periphery is adherent to the cornea. There is some increase of nuclei in the sclerotic and cornea, especially in the episcleral tissue and in the walls of its blood-vessels. There is great excess of nuclei in the O. N. and some increase in its connective tissue. | The sclerotic is very thin and staphylomatous at the outer equator, where it is nearly perforated by the tumour. | .. .. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 419. George B., æt. 49, under care of Mr. Tay. Left eye painful for 2 years. *At excision*, no *p.l.* There is a large prominent staphyloma of the upper ciliary region, with ciliary thinning elsewhere. | Tension increased. Arteries of ciliary region not recognisable. | The retina is detached and with the lens has almost disappeared from recognition. A tumour, the size of a hazel-nut, projects from the choroid at the outer side of the eye, overhanging the O. D. It has a narrow neck, and then shelves gradually by a broader base into the unaffected choroid, extending in front beyond the equator. It is made of cells whose body is formed by an oval staining nucleus, in which is a dot-like nucleolus. The extremities of the cell are drawn into short tails. The blood-vessels of the tumour are thin-walled and not very numerous. The relation of the tumour to the choroidal layers cannot be traced with any certainty. | The fibrous trabeculæ of the O. N. are hypertrophied. They contain a slight excess of nuclei. The nuclei of the nerve-bundles are also increased; some are very like tumour cells in size and shape; they contain, however, in place of a dot-like nucleolus, several fine granules. The ciliary body and iris are much shrunken. They contain a considerable excess of nuclei and but very little muscular structure. The pigment epithelium layer of the P. C. R. consists, apparently, of elongated brown pigment cells disposed loosely at slightly different levels, with a fibrillated nucleated tissue between them. It is, on the whole, considerably less darkly pigmented than usual. The fibres of the zonula are hypertrophied and contain an excess of | .. .. | 18 months later there was no recurrence. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 419.—*continued.* | | | nuclei. The ciliary folds are excessively thin, much extended forwards, and are in close contact with the iris, which is adherent to the cornea. There is nuclear increase both in sclerotic and cornea, and more especially in the walls of the blood-vessels of the episcleral tissue. | | |
| No. 367. Clara W., æt. 23. Left eye dim 8 months, pain 6 weeks. Some of the points in this case have been already briefly reported in its relations to glaucoma, in vol. ix, p. 401. | Tension increased (T. 1). Arteries of ciliary body much enlarged, as also the long ciliary arteries. | There is a sarcoma the size of a small bean at the lower and inner quadrant of the globe, reaching from just in front of the equator to the O.D. and just extending round this into the choroid on its opposite side. The tumour is smooth and very slightly pigmented. It is permeated by large and numerous thin-walled blood-vessels. It is made up of fusiform nucleated cells. The retina is detached and semi-contracted. | The ciliary muscle is somewhat atrophied and a little inflamed. The iris also is a little inflamed, its peripheral part is applied to the cornea. The ciliary folds are not much shrunken, but far removed from the iris. There is considerable increase of nuclei in the optic nerve trunk, probably due to a neuritis. | There appears to be a general slight increase of nuclei in the choroid. | .. .. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 240. Patient under care of Mr. Lawson (not from Moorfields Hospital). Globe much enlarged, with great ciliary thinning, especially at upper part. Cornea leucomatous at lower part, and to this the iris and lens are adherent. Retina detached. | Tension probably much increased. | An unpigmented tumour the size of a hazel nut overhangs the O. D., and appears to spring from it by a narrow pedicle. In reality, however, its connections are with the adjacent choroid. It consists of round or slightly oval cells with dot-like nuclei, and contains numerous thin-walled blood-vessels. The tumour cells are in immediate relation with these. | The O. N. has its trabeculæ hypertrophied. Bodies resembling tumour cells are scattered evenly in the nerve bundles, which contain no nuclei other than these. The sclerotic contains an excess of evenly-distributed large nuclei, which, however, are especially abundant in the walls of its blood-vessels. | .. .. | .. .. |
| No. 497. George W., æt. 43, sailor, under care of Mr. Tweedy. He noticed nothing wrong till 9 weeks ago, when he had severe pain in his *Right* eye during the night. Next morning he found he had no *p.l.* with it. The pain | Tension much diminished at excision. Arteries of ciliary body are very small. | The retina is detached and scarcely recognizable except near the O. D., where it presents an occasional brown pigment mass in its inner layers, but is otherwise quite unaffected. The choroid below is thickened into a dark coloured mass which extends from the lower ciliary region, which it involves right up to the lens, to the O. D. which it | The ciliary body is shrunken and converted into fibrous tissue, which contains scarcely a trace of muscular structure. The pigment epithelial layer of the P. C. R. is represented by several very imperfect layers of isolated brown masses, between and internal to which is a layer of imperfectly fibrillated nuclear tissue. The iris is | .. .. | .. .. |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 497.—*continued.* | | | | | |
| lasted 2 days, and since then he has had only slight pain occasionally. *At excision*, cornea clear. A.C. shallow, lens advanced near the cornea, pupil large, iris discoloured and dark, especially at its lower part. | .. .. | overhangs. Its anterior part is 6 mm. thick, and the tumour cells even extend into the adjacent peripheral part of the iris. Microscopically, it is made up of rather small cells of round or slightly oval shape. They stain well and contain no nucleus, but many fine granules. They have no unstaining peripheral zone. There is no stroma other than the numerous very large, thin-walled, blood-vessels. In the tumour there are occasional tracts which scarcely stain at all and are made principally of large pale-brown pigment masses with an ill-defined intermediate substance. These are probably the seats of previous hæmorrhages. There are occasional more darkly pigmented tracts, especially | atrophied and its peripheral part adherent to the cornea. There is considerable excess of nuclei in the cornea and sclerotic, and more still in the episcleral tissue, especially in the walls of its blood-vessels. The nuclei of the sclerotic in the neighbourhood of the tumour can be traced in all gradations from ordinary connective tissue nuclei into bodies very like the unpigmented cells of the tumour. In the inner scleral layers at this part there are many pigmented cells. The optic nerve has its fibrous trabeculæ hypertrophied and very vascular. There is an increased number of nuclei both here and among the nerve-fibres. All gradations are presented from connective tissue nuclei to tumour cells. | | |

B.—*Intra-ocular Tumours.* (*a*) *Sarcoma of the Uveal Tract*—continued.

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| No. 497.—*continued.* | | near the sclerotic. At the edge of the tumour its cells extend into all the layers of the choroid though less far into the chorio-capillaris than the rest. | All are pretty evenly scattered. | | |
| No. 225. Charles T., æt. 28, sailor, under care of Mr. Hutchinson. Dimness of *Left* eye noticed 7 months ago. Three months after that sudden pain came in it during the night. He had then scarcely *p.l.* with it. Two weeks later the lower part of the cornea became ulcerated. *At excision* there was leucoma of cornea consequent | Minus tension at excision. The arteries of the ciliary body are very small. | There is a sarcoma the size of a pea growing from the choroid at the inner equator. Its posterior part does not extend to within 4 mm. of the O. D. Its anterior part overhangs the ciliary body but does not reach the lens. It rises rather abruptly at its margin from the choroid, which is somewhat thickened by an excess of very dark pigment cells in its outer layers and by similar cells mixed with smaller, unpigmented, roundish, deeply-staining cells in its middle layers. The chorio-capillaris is very | The O. D. is much swollen. There is a great increase in the number of staining nuclear bodies in the O. N. These present all shapes intermediate between the elongated connective tissue corpuscles of fibrous tissue and bodies very like the unpigmented cells of the tumour. The structure of the ciliary body and iris, especially the muscular fibres, is much atrophied, but neither shrunken up nor sclerosed. The apices of the ciliary folds are adherent firmly to the lens capsule. They are not | The retina is *in situ*. The vitreous is yellowish in tinge, but otherwise normal. There is nuclear increase in the sclerotic and cornea, especially in the walls of the blood-vessels and in the episcleral tissue generally. Some of these last are very | Three years later there was no recurrence. |

B.—*Intra-ocular Tumours.* (*a.*) *Sarcoma of the Uveal Tract*—continued.

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| No. 225.—*continued.* | | | | | |
| on the ulceration. | .. .. | little affected, and can be traced over the greater part of the tumour as a thin layer. The tumour itself has the pigment cells in excess in its outer layers, and the small unpigmented cells more abundantly in its internal part. These last are generally oval and contain fine granules. There are among them numerous thin-walled blood-vessels. The uninvolved choroid is thickened by extreme tortuosity and enlargement of the blood-vessels of its middle and outer layers. There is also a great increase of darkly pigmented stellate cells in the same layer. | shrunken but still quite clearly separated by an interval from the periphery of the iris, which is adherent to the cornea. The posterior surface of the more central part of the iris is adherent to the lens capsule. There is an excess of stellate brown pigment cells among the remains of the muscular fibres of the ciliary body and in the iris. The pigment from the epithelium of the anterior part of the P. C. R. is collected in rounded lumps, which are scattered irregularly in the subhyaloid space. | like the tumour cells. | |

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| No. 307. Richard E., æt. 5, under care of Mr. Adams. *Left* eye noticed for 2 months as presenting a white reflex. Ant. chamber of moderate depth. Pupil dilated. The iris is flat and the lens *in situ*. | Tension increased. Arteries dilated. | Posterior part of tumour forms a solid mass of glioma cells projecting from O.D. In the anterior part of globe the detached retina is recognizable; its folds as seen in section are occasionally thickened into the tumour. The thickenings often appear as if due to a proliferation of the inner granules. Sometimes, however, the thickening is found most distinctly in the outer granules alone. It often extends from the inner granules into the nerve cell and fibre layers. Each blood-vessel of the tumour has a space round it, as if it had a lymph-sheath space free from tumour cells. Near the O.D. zones of cells deeply staining with logwood surround the blood-vessels, whereas, those further removed from them stain less. | The nerve fibres in the centre of the papilla are replaced by glioma cells, which extend backwards as far as the lamina cribrosa. The O.N. is free from the tumour, but there is a little neuritis. The choroid is free. The ciliary body is much inflamed, but not atrophied. The iris is a little inflamed; its peripheral part is a little atrophied where it is adherent to the cornea. The ciliary folds are of fair size, and somewhat advanced, so as to be in some specimens very near to the posterior surface of the peripheral part of the iris. The lig. pectinatum and neighbourhood of the canal of Schlemm are rather inflamed. The first is shrunken and partly sclerosed. | The cornea and sclerotic have yielded to pressure from within the eye, both in front and behind the sclero-corneal junction, which retains its position. Their nuclei are decidedly increased, especially in the episcleral tissue and in the walls of its blood-vessels. | The tumour recurred, and the contents of the orbit together with the enormously thickened lids were removed. Chloride of zinc paste was afterwards used. Two months later the tumour had recurred a second time and grown to a very large size. The child died shortly afterwards. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 257. Bertie H., æt. 2½, under care of Mr. Hulke. A white reflex was noticed from his *Left* eye 9 months ago. *At excision*, his cornea was rather prominent; his pupil widely dilated, with a reddish-yellow mass visible through it. | Tension increased. Arteries dilated. | A large solid mass of tumour cells project from the O.D. The anterior part of the retina is not involved in the tumour. The choroid on either side of the O.D. is vastly thickened; its structure is destroyed and replaced by glioma cells. Each vessel of the tumour, whether an artery, capillary, or vein, has surrounding it a loose zone of connective tissue, apparently its sheath. This is quite free from tumour cells. | The optic nerve is very short, but, as far as it goes, its nerve bundles are entirely replaced by glioma cells. In it are longitudinal streaks of a lighter coloured tissue, which appears to be fibrillated imperfectly. The ciliary body is very little atrophied, but considerably inflamed. The iris periphery is much atrophied where it adheres to the cornea: elsewhere, the iris is much inflamed, but not atrophied. The ciliary folds are advanced, so as to be in some specimens close to the iris. | The sclerotic cornea and scleral-corneal junction are as in the case above, 307. The globe is nearly full. The ant. part of the retina is detached. | The tumour recurred in the orbit. |
| No. 258. James W., æt. 6½, under care of Mr. Couper. Nothing was wrong as far as known till 7 months ago, when he received a blow on his *Left* eye, | Tension has undoubtedly been increased. No information as to ciliary arteries. | The tumour fills the globe. Its anterior part is semi-fluid. Its posterior part projects as a solid mass from O.D., and in it no retinal structures can be recognized. Tracts of fibrous tissue extend into it | The deeply staining glioma cells only extend into the superficial layers of the lamina cribrosa. The cells found more deeply stain but faintly with logwood. They are found between the hypertrophied fibrous | The ciliary body was not examined microscopically. The iris was in contact with the cornea. | The child died with cerebral symptoms 1 month later. The eye was so tender that it was difficult to esti- |

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| No. 258—*continued.* | | | | | |
| which however appeared to cause no structural lesion. Two weeks later his father noticed that the child could not see with that eye. *At excision* the upper ciliary region was very staphylomatous. | | from the O.D. as well as blood-vessels. Each blood-vessel has a distinct and large sheath space free from tumour cells. The glioma contains many hæmorrhages and calcareous particles, which are visible to the naked eye. | trabeculæ of the nerve, taking the place of the nerve-bundles. The central artery is flattened; its endothelium is a little proliferated. The vein is not recognizable. The connective tissue ensheathing them is free from tumour. The inner and outer sheaths of the O. N. are fused together. They are much thickened; their fibres are separated somewhat, and between them are sometimes found glioma cells, which stain very faintly. The diameter of the nerve and its sheath is much increased. | | mate the tension. The patient, as well as the other 3 children of the family, looked strong and well-nourished. |
| No. 162. Charlotte W., æt. 4, under care of Mr. Streatfeild. The parents have noticed a white reflex from | Tension increased. Ciliary arteries dilated. They are filled with a structure- | The tumour projects from the O.D. as a solid mass of glioma cells, which contain tracts of fibrous tissue and blood-vessels provided with sheaths. As we pass for- | The glioma cells are sparse among the fibres of the lamina cribrosa, but behind it they entirely replace the nerve fibres, forming solid columns, se- | The tumour recurred in the orbit, apparently in the optic nerve stump. | The uniformly-distributed nuclear bodies of the deeper parts of the nerve |

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| No. 162—*continued.* | | | | | |
| the eye for 6 months. | less substance in which some bodies like white blood - corpuscles are observed. | wards the retina gradually emerges from the tumour, and its more anterior part, though detached, is unaffected, except that isolated tracts of glioma cells are found in its nerve fibre layer, thickening the retina. The centre of each tract is seen to be a small artery, which is surrounded by its sheath space, and then by the cells. The choroid is invaded by glioma cells only at the upper and inner part of the globe. In front some cells are seen on its internal surface. | parated from each other by the fibrous trabeculæ of the nerves. Farther away from the eye these columns end abruptly, though not quite at the same level. Beyond them, nuclear bodies somewhat like glioma cells, or like enlarged connective tissue nuclei are abundantly and uniformly distributed among the nerve fibres for some distance. The cut end of the nerve is in this condition. The arteria centralis and its sheath are perfectly free throughout. The ciliary body is inflamed and much atrophied. The ciliary folds are much atrophied, but extend to a considerable distance directly inwards. The iris is inflamed and its periphery adherent to | The cells of the mass were traversed in places by fibrous bands. Elsewhere they were spreading between fat-cells. | might have passed for evidences of neuritis, except that they were too large, and that the tumour recurred after the nerve had been divided here. They might well pass for glioma cells spreading from the tumour, except for their uniform distribution, both near and farther away. Might they be neuroglial corpuscles taking on the cha- |

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| No. 162—*continued.* | | | the cornea. The pars ciliaris retinæ has no tumour cells on its internal surface. | | racters of glioma cells? |
| No. 256. George G., æt. 5. He has had a cast in his *Left* eye since birth, and 6 months ago his parents noticed a white reflex from it. *At excision* he has had pain and inflammation in it for 3 weeks. | Tension increased. Arteries of ciliary body enlarged. | The entire lower part of vitreous cavity is filled with tumour cells. The upper and outer part is more free but still dusty, with scattered cells. Upper and outer part of retina free. In a horizontal section through the O. D. one limb of the retina is entirely converted into deeply staining glioma cells, whereas the other limb, though detached, is entirely free with the exception of a little thickening of both layers of granules close to O. D. The tumour presents the usual characters. All its blood-vessels have distinct sheaths. The choroid is free from tumour cells. | The tumour cells extend a little way into the optic disc, but do not reach the lamina cribrosa. Beyond this the fibrous trabeculæ of the nerve contain more and larger nuclear staining bodies than normally. This may be due to some slight neuritis. The O. N. was cut rather close to the globe in the excision. The ciliary body and iris are much inflamed, but not atrophied. The most anterior ciliary folds are directed forward so as to be almost in contact with the periphery of the iris, which in its turn is adherent to the cornea. Numerous tumour cells are on the pars ciliaris retinæ, | There is much inflammation of the loose episcleral tissue and its blood-vessels. | The patient was seen 1½ years later, and there was then no recurrence. The slight excess of nuclei in the nerve was probably due to neuritis only. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 256.—*continued.* | | | especially between the ciliary folds, and some lie in the ligamentum pectinatum. Many of the ciliary folds are directed directly inwards. | | |
| No. 420. Arthur F., æt. 2½ (*Right* eye excised during the temporary absence of the Curator). Ant. chamber normal. Globe apparently full of glioma mass. Under care of Mr. Wordsworth. | No note of tension. Arteries of ciliary body are somewhat enlarged. | The globe is full of a soft glioma mass which projects from its attachment at the O. D. The retina is not recognizable in the posterior part of the globe though its front part and the pars ciliaris are quite unaffected. The choroid is free from tumour cells. On either side of the O. D. the sclerotic has yielded somewhat to pressure from within the globe. | The tumour cells extend into the O. N. as far as its cut end which is not far removed from the eye. They form linear tracts separated by the hypertrophied fibrous trabeculæ, especially near the circumference of the nerve. The ciliary muscle and iris are inflamed but not atrophied. The iris periphery is adherent to the cornea. The ciliary processes are adherent to the cornea. The ciliary processes are advanced and in some instances are applied to the adjacent posterior surface of the iris. | The lens is *in situ*. The cornea and sclerotic have yielded to the intra-ocular pressure both in front of and behind the sclero-corneal junction. The corneal and scleral tissue is inflamed. | When seen 4 months later a firm rounded mass could be felt under the upper lobe of the conjunctiva. It rapidly increased in size. The lump was first noticed by the parents a month before. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 130. Florence G., æt. 2 months, under care of Mr. Streatfeild. The parents noticed something wrong with the child's *Right* eye 3 weeks ago. There is now a reddish-yellow reflex from behind lens. The pupil is of medium size. The ant. ch. is shallow. | Tension increased. Circulus arteriosus much enlarged. | A mass of glioma cells projects from the O. D., from which arteries are seen passing into it. No retinal structures can be recognized here, but the anterior part of the retina is but little changed in structure, though it is detached and folded forward on the pars ciliaris retinæ. At a little distance behind the ora serrata the tumour begins as if a thickening of the outer granules (Glioma Exophytum. Hirschberg). The inner granules become also affected, and a little further back the nerve cell and fibre layers are much thickened by tumour cells. A little further back all traces of retinal structure are lost. The globe is filled by the soft yellowish glioma mass which contains numerous calcareous particles. | The tumour cells extend into the nerve as far as the lamina cribrosa, and a few are found among the most superficial fibres of this. It is much pushed backwards. Behind it no tumour cells are recognized for certainty in the optic nerve, though large nuclear bodies are distributed through it more abundantly than in the normal state. These may, however, be the result of a neuritis. The ciliary body and iris are much inflamed but not atrophied. The iris periphery is applied to the cornea. The ciliary processes are considerably advanced. Nodules of glioma cells are seen on the pigment epithelial cells on the vitreous lamina of the choroid near the O. D., but otherwise the choroid is free. | The cornea and ciliary sclerotic have yielded more than that at the sclero-corneal junction, which thus appears as if pulled in. The cornea and sclerotic are somewhat inflamed. | 13 months later there was a yellowish vascular reflex from the *Left* eye which was blind. The child seemed still healthy and well nourished. 2½ years later the *Left* was excised (*see* No. 542 below). There was no return in the *Right* socket. The other children of the family appeared strong and healthy. |

B.—*Intra-ocular Tnmours.* (*b*) *Glioma of the Retina*—continued.

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| No. 542. The left eye of Florence G. (*see* above). This was excised 2½ years later than the other. | Cornea perforated and tension now diminished. Ciliary arterial circle not recognized in the much atrophied ciliary body. | The tumour cells fill the globe. No trace of retinal structures is found anywhere. The choroid cannot be recognized at all at the posterior part of the globe. Tracts of deeply staining tumour cells surround the blood-vessels, but the intervening cells stain very little and are of very faint outline. The cells near the sclerotic are the most deeply staining of all. The pars ciliaris retinæ is unaffected. | The cells extend from the globe into the optic nerve for a little distance, but soon they become replaced by a nearly structureless, very slightly fibrillated, substance which fills up the gaps between the hypertrophied fibrous trabeculæ of the nerve. The nerve sheaths have their fibrous tissues enormously hypertrophied. The intersheath space can only be recognized by the cells like those of glioma, which are found tolerably abundantly here. The ciliary sclerotic and cornea are much stretched and thinned. The ciliary body is vastly thinned. Its structure is more fibrous than muscular. Remains of an inflammatory exudation are found on its internal surface. The iris is much thickened by occasional nodules of glioma cells in its structure, and | There is an uniform staphylomatous bulging of ciliary cornea and sclerotic. The cornea and episcleral tissue contain abundant deeply staining nuclear bodies. Similar bodies are found in the sclerotic and in the walls of its blood-vessels. | It is interesting to note the condition of the optic nerve with regard to the question of transmission of the tumour from one eye to the other. Note also that there was no recurrence in the socket of the Right eye. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 542—*continued.* | | | also by a tough fibrous exudation on and in its posterior wall, in which the remains of the lens capsule are embedded. It is adherent to the cornea. | |
| No. 343. Eleanor P., æt. 3, under care of Mr. Adams. The parents have noticed something wrong with the *Left* eye for 7 months. The child is a little pallid, but well nourished. | Tension 2. Ciliary arteries dilated. | A solid mass, composed of cells with penetrating bands of fibrous tissue and blood-vessels with sheaths, projects from O. D. As we pass forwards it soon resolves itself into the much thickened retina. The thickening is distinctly due to an infiltration of tumour cells into the nerve cell and fibre layers, for the granule layers are undoubtedly unchanged in many places. The choroid is free from tumour cells. | The tumour cells do not extend back in the optic nerve beyond the lamina cribrosa. The nerve presents an increase of the nuclei of its trabecular tissue, but is otherwise healthy. The ciliary body and iris are a little inflamed, but not atrophied. The iris periphery is applied to the cornea. The ciliary folds are covered with glioma cells. They are rather attenuated and somewhat advanced, so as to be close to the posterior surface of the adjacent *iris*. | The remains of the vitreous lie behind the lens, and occupy only a fifth part of the normal space. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 539. Thomas C., æt. $3\frac{2}{3}$, under care of Mr. Lawson. His parents noticed a white reflex from his *Right* eye 22 months ago. It became gradually more conspicuous till 6 months ago, when the pupil was dilated. Since then he has had pain. Now cornea clear except for some glioma cells on its posterior surface. The ant. chamber is extremely deep and wide. Lens semi-opaque. Much ciliary thinning, especially above. | Tension 2. The ant. part of the ciliary body is invaded by tumour cells, and its artery cannot be recognized. | The tumour extends forwards from O. D. as a band composed of blood-vessels, a little fibrous tissue, and glioma cells. Its narrowness appears to be due to the compression exercised by the much thickened choroid on either side. Anterior to this the glioma cells fill the entire vitreous cavity up to the posterior surface of the lens. Among them are many calcareous particles. The most anterior part of the retina is unaffected. It is folded forwards on the ciliary body, though separated from this in front by tumour cells. The choroid is vastly thickened by tumour cells, which have destroyed its proper structure. | The glioma cells extend back into the nerve very abundantly as far as the lamina cribrosa. In this they are more sparsely mixed with its fibrous tissue. Behind it they are again more abundant, and they only terminate just in front of the cut end of the nerve. Close up to this there are scattered a moderate number of staining bodies, which may be tumour cells. The ciliary body is somewhat inflamed and atrophied. Glioma cells are found in the connective tissue just below its internal surface, and abundantly over the region of the pars ciliaris retinæ, which cannot itself be recognized. The ciliary folds are attenuated and much stretched forwards. They are, however, far removed from the peripheral part of the iris and cornea by | The globe is enlarged in all its diameters. The anterior chamber of the aqueous is enormously deep and wide. | The patient looks very healthy and well-nourished. The tumour recurred shortly in the socket with a fatal result. The *Left* eye also became gliomatous. He died in convulsions. |

B.—*Intra-ocular Tumours.* (*b*) *Glioma of the Retina*—continued.

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| No. 539—*continued.* | | | reason of the staphyloma of the latter. There is a staphylomatous thinning and bulging of the margin of the cornea, with which the remains of the atrophied iris periphery have coalesced. Tumour cells are found on the pigment of the posterior surface of the iris and in its tissue close to its anterior surface. Hence they extend into the adjacent corneal lamellæ. Glioma cells are also found on Descemet's membrane. | | |

## CURATOR'S PATHOLOGICAL REPORT—continued.

*Two Cases of Glaucoma of extremely short duration.*

THE microscopical preparations were made by Dr. L. Webster Fox.

601. Charles F——, æt. 37. Under care of Mr. Tay. Six days before excision a fragment of metal penetrated the cornea and wounded the lens of his left eye. Three days later, when he came as out-patient, the tension was increased and the anterior chamber scarcely existed. After three more days there was severe pain with T 3: the eye was then excised, but no foreign body was found.

The eye generally was healthy though the lens was opaque, rather soft, thinned from before backwards and bulged forwards so as to fit closely to the posterior surface of the iris which was applied to the cornea. There was considerable excess of nuclei in iris and ciliary body, especially about their muscular tissue. The fibres of ciliary muscle were lengthened in a meridional direction and decidedly attenuated. The ciliary folds were small and displaced backward: arteries of ciliary body somewhat enlarged. Canal of Schlemm and episcleral tissue moderately inflamed.

The optic nerve has a decided increase of nuclei in its connective tissue. The disc is not cupped. There is no excess of nuclei on its vitreous surface. The choroid is not inflamed, but is rather thin and atrophic.

616. Robert S——, æt. 44, under care of Mr. Tweedy. Seven weeks before excision he came complaining that the sight of his left eye had been failing for a year. The retina was found detached at the lower and outer part, but he still read Jaeger 20. Pupil large and sluggish: tension diminished. Ten days before excision, after a violent attack of pain during the night, he found he could not see light. Three days later his eye was tender with T.n., but still no *p. l.* The next day there was great pain with increased tension for which an iridectomy was performed three days later. Still the tension recurred with great pain necessitating excision.

*At excision:* The retina was detached in an umbrella form.

A pedunculated tumour the size of a large pea sprung from the lower and outer part of the choroid, which it involved by a flattened base nearly as far as the ora serrata in front and to within 4 mm. of the papilla behind. Its cells are spindle-shaped, its vessels are numerous, large, and thin-walled. The uninvolved chorio-capillaris can be traced over nearly the whole internal surface of the tumour. Similar cells are found in the outer layers of the adjacent choroid. They do not, however, reach so far as the optic disc, the intervening choroid being compressed and atrophied, though its vessels are full of blood corpuscles.

The episcleral tissue corresponding to the tumour is much thickened and inflamed, and this condition extends all the way, though in a less degree, backwards to the optic nerve and forwards to the ciliary region.

The iris and ciliary body at parts removed from the operation are considerably inflamed, but not atrophied. The iris periphery is applied to the cornea; the anterior ends of the enlarged ciliary folds are in contact with it in places. The arteries of the ciliary body are enlarged. The lig. pectinatum is inflamed. Inflammation can be traced along vessels extending between the inflamed episcleral tissue and the ciliary body

## CURATOR'S PATHOLOGICAL REPORT—continued.

### *Seven Cases of Suppurative Hyalitis.*

THE following cases have been selected as being fairly illustrative of the phenomena of pus-formation in its various stages:—

No. 513. Flora D——, æt. 7, under the care of Mr. Lawson. Two weeks ago she received a blow which caused a penetrating wound of sclerotic just below the corneal margin. At excision T 1. *Cornea* clear. Iritis many pointed posterior synechiæ: no hypopyon. *Vitreous:* posterior part is thin and fluid and purulent. Anterior part more consistent and purulent. *Sclerotic* inflamed. This change extends backwards from its ciliary part to the optic nerve, and is continuous with the neuritis there.

*Ciliary body.* Arteries enlarged. Ciliary muscle is of fair size and shape, its bundles thickened with far more nuclei than usual on them and also scattered between them; most of them are elongated. A few multi-nuclear corpuscles are scattered equally over the connective tissue of the ciliary body, but not more towards its pigment epithelium surface, but rather less.

P.C.R. Columnar elements vastly elongated except at extreme front part: their nuclei are always large, oval, and pale. The pigment on which their external ends rest is a little increased, but not much. Among these columnar elements, but distinct therefrom, are vast numbers of smaller deeply staining multi-nucleated bodies, continuous internally though not equally thickly into the vitreous: externally they go right up to the pigment cells but not beyond them, the connective tissue of the ciliary body being perfectly free therefrom.

Internal to the P.C.R. is a stratum somewhat fibrillated concentrically in which cells are found, some exactly like vitreous corpuscles, others similar but caudate.

Next follows a broad zone consisting of a fine reticulum produced probably by the coagulation of some albuminous substance; within its meshes deeply staining multi-nuclear cells (2, 3, or 4 nuclei) lie scattered. Still more internally, and occupying the vitreous centre, is a mass consisting of large cells,

most of which have a large feebly staining nucleus. The cell outline is ill-defined and their substance opaque and finely granular (diam. = ·024 mm.). Scattered among these latter are a few others (about 2 per cent.) resembling closely the smaller cells above described within the reticulum.

*Optic disc* much swollen: in and round walls of arteries of papilla are numerous uninuclear bodies. On the retinal surface of O.D. is a mass of cells, most of which are like vitreous corpuscles: some tend to fibrillation. There are also some smaller multi-nucleated bodies as in centre of vitreous. There are no multi-nucleated bodies in the walls of the inflamed blood-vessels of the papilla or in the optic nerve.

*Choroid:* round undivided nuclei are found in scattered groups in a few places.

*Retina:* nerve fibre layer much thickened with nuclear bodies of which a few are multi-nucleated.

*Blood-vessels* of the eye generally contain an excess of colourless blood corpuscles which are multi-nucleated.

NOTE.—All measurements and observations were made on preparations which had been hardened in Müller's fluid and alcohol.

No. 204. William B——, æt. 60. A cataractous lens was extracted 4 months ago. Though there appears to have been some slight iritis the eye did well generally till 2 months later, when increased tension came on. Iridectomy upwards was done and repeated downwards 6 weeks later, but without good result. After the second operation the vitreous suppurated.

Tension somewhat in excess. The arteries of the ciliary body are enlarged.

The iris is adherent to the scar of the wound at the edges of the iridectomy gap. There is a large excess of nuclei in the episcleral tissue, and a moderate excess in the corneal scars. The adjacent iris contains dense groups of nuclei at various points. The ciliary muscle is much inflamed as well as the connective tissue among its fibres and on its internal surface. The corpuscles found among the most external muscular layers are often multi-nucleated, while those in the internal layers have almost universally one nucleus alone. The pigment layer on the internal surface of the ciliary body has many multi-

nucleated cells visible among its pigment granules. The columnar elements of the P.C.R. are elongated and contain between them a very large number of multi-nucleated corpuscles. The vitreous zone internal to the P.C.R. contains comparatively few cell-elements distributed in a loose structureless reticulum. They are nearly always like vitreous cells, *i.e.*, large nucleated cells, though among them are scattered a few multi-nucleated bodies, like those among the columnar elements of the P.C.R. The vitreous in its more central portions consist of cells with no intervening tissue. They are large and mostly of very faint outline with a scarcely recognizable nucleus, though some are more distinct in outline, and have a large and distinct nucleus. Also there are a few smaller and multinuclear.

The O.D. is swollen much; there is great inflammation of the nerve trunk; in its anterior part the increase of nuclei predominates while in its posterior part there is considerable increase in its connective tissue. None of the elements found here show direct indications of multiplication by division of nuclei. The walls of the arteries of the papilla are thickened as if by inflammation, and there is a larger number of endothelial elements in them than normal. The choroid is thicker than usual, apparently from œdema; its nuclei are more abundant than normal at a few points only. Its blood-vessels are distended with red blood-corpuscles. The retina contains some excess of nuclei, especially in its nervous layers in the neighbourhood of the O.D. Its posterior part contains many rounded masses of nuclei, each corresponding apparently to the blocked lumen of a blood-vessel. Its anterior part is unaffected. Many cells, just like vitreous cells in excessive number, and some rather smaller and multi-nucleated, remain attached to the vitreous surface of the O.D.

The blood-vessels of eye generally contain white blood-corpuscles in excess, and these are multi-nucleated.

The sclerotic presents a general slight nuclear increase, especially in the blood-vessels of its most external layers, and the loose tissue over them.

No. 215. Robert R——, æt. 30, under care of Mr. Couper, had

gonorrhœal ophthalmia of *left* eye 8 years ago, which left the cornea leucomatous. Inflammation came on spontaneously 5 days before excision and was diagnosed as suppurative panophthalmitis. T. 1½.

The *vitreous* greenish-white and opaque at centre in front; greenish and translucent behind: internal to ciliary processes there is a translucent layer, while on the ciliary processes themselves there is a purulent deposit.

*Microscopically*, the *iris* contains a considerable number of multi-nuclear with a still greater proportion of uninuclear corpuscles. The exudation in front of iris is made up of many multi-nuclear bodies in a scanty reticulum like coagulated albumen.

The *ciliary body* is infiltrated with nuclei; many are elongated, and like those of its muscular fibres; many are in its connective tissue, and especially in that near its internal surface: the vast majority of them are uninuclear, while very few are multi-nuclear.

*P.C.R.:* its pigment layer is a little irregular; it is separated from its basement membrane by an irregular layer of nuclei: Its *columnar elements* are scarcely recognizable, being obscured by many multi-nuclear bodies with a few pale pigment globes and scattered pigment granules. Then follows internally a zone, separated by a well-defined band (the hyaloid) from the columnar elements, and which consists of a reticulum and corpuscles, some large and uninuclear and quite like vitreous cells, others smaller on the average, and multi-nuclear. The central part of vitreous is made up entirely of cell elements, most of which are large, with a faint outline and uninuclear, others somewhat similar in appearance, but rather small, and multi-nuclear.

The *optic disc* is swollen: there are nuclei round its arteries, in their walls, and in the connective tissue of the optic nerve.

*Choroid* shows a general increase of uninuclear bodies which are not in especial relation with the blood-vessels: it is otherwise normal. There is no increase of colourless blood-corpuscles within the blood-vessels, but a considerable proportion of these visible are multi-nuclear.

The *retina* is slightly œdematous and contains a slight excess of multi-nuclear corpuscles, especially in its nervous layers,

and notably in the walls of its arteries. On its internal surface is a thin imperfect layer, some of whose cells are exactly like vitreous corpuscles and uninuclear, while others are similar but multi-nuclear; others, again, are the same, but smaller still.

*The blood-vessels of eye* contain some excess of white blood-corpuscles, which are multi-nuclear.

No. 510. Walter P——, æt. 44, under care of Mr. Tay. An accident to his eye 30 years ago appears to have been followed by iritis, for it has been dim ever since, though not otherwise troublesome. Acute spontaneous inflammation arose 14 days before the excision. T.n., or slightly increased.

The *vitreous* is opaque and purulent in its anterior central part; its posterior part is clearer. A much less opaque zone separates it from the ciliary processes, which are covered with a pus-like layer. The *optic disc* is swollen, its veins are very full and tortuous.

*Ciliary body:* the nuclei of its muscular fibres are increased in number. An increased number of uninuclear bodies is found in the connective tissue between them. The blood-vessels contain excess of proliferated endothelial (uninucleated) cells. Within them is slight excess of white blood-corpuscles, which contain 3 or 4 nuclei grouped together.

*P.C.R.:* its pigment epithelium layer is more highly pigmented and distributed over a larger area than normal: a layer of nucleated bodies (most of which have multiple nuclei) separates it from its basement membrane. Numerous similar bodies can be faintly seen among its pigment granules. Its *columnar* elements are elongated; numerous nuclear bodies (multi-nucleated) are found between them and on their internal surface in the sub-hyaloid space. The *hyaloid* is distinctly seen: internal to it is a broad zone, made of a fine reticulum as if of some coagulated albuminoid substance, containing but few cells (not 1 per cent. of those in the adjacent sub-hyaloid space and among columnar elements of P.C.R.): occasional ones are large and caudate, and some like ordinary vitreous corpuscles, whereas others are smaller and multi-nuclear. All are somewhat larger than typical white blood-corpuscles. Towards the vitreous centre is a solid mass of cells; all are large, with very indistinct

outline, and usually a single and faint nucleus, but some are multi-nucleated. No cells are so small as some of those described in vitreous near P.C.R.

*Optic disc:* Great excess of nuclei in walls of blood-vessels of papilla and in the loose tissue round them: great excess of nuclei in connective tissue of optic nerve. All, whether in the connective tissue or in walls of blood-vessels, are uninuclear. On the vitreous surface of optic disc is a cluster of nucleated bodies, some of which are large and distinctly vitreous cells; many are somewhat smaller and multi-nucleated. These are exactly similar to those near the vitreous centre, but a trifle smaller.

*Choroid* is free from nuclear increase generally, but there are some clusters of nuclear bodies in its middle layer near the optic disc.

*Retina* presents slight increase of nuclei in its nervous layer near optic disc, especially in walls of its arteries.

White blood-corpuscles are in excess in the blood-vessels and are multi-nuclear.

No. 191. Thomas S——, æt. 31, under the care of Mr. Tay. A fragment of metal traversed the cornea of his *right* eye 5 weeks before excision, and was subsequently found lying in front of the lower part of the vitreous cavity. *At excision:* T + : Iritis with extensive posterior synechiæ. Anterior chamber very shallow: lens cataractous: the detached retina extends from optic disc to ora serrata and is semi-contracted: an opaque yellow purulent substance lies in the front part of the cavity and represents the shrunken and suppurated vitreous.

The ciliary folds and lens are advanced and in close opposition with the iris. The iris-periphery is applied to the cornea. The ciliary arteries are enlarged.

*Microscopical appearances: P.C.R.* The columnar elements are much elongated in places, and their characteristic pale large oval nuclei are numerous and disposed at different levels (the Müllerian fibres of adjacent retina are also extremely elongated). Between them is a moderate number of small multi-nuclear bodies. The *pigment* granules of its pigment epithelium are black and scattered, especially towards its posterior part: among

them are evidences of numerous partly obscured multi-nuclear cells: occasional tongue-like processes of scattered pigment granules mixed with numerous similar multi-nuclear cells extend inwards from pigment epithelium of P.C.R. towards the cavity of the globe, raising up or extending between the columnar elements of the P.C.R.

*Ciliary muscle:* fibres inflamed; a number of nuclei are scattered between them (and also in iris), but extremely few bodies with multiple nuclei.

The shrunken *vitreous* is traversed by a fibrillation radiating towards the centre of its anterior part. Spindle-shaped and elongated nuclei are disposed at short intervals along the course of this fibrillation. Among them are a few blood-vessels running out from the ciliary body. Intertwined with this stroma are corpuscles much smaller on the average than the vitreous cells, many of them being multi-nucleated. Some few, however, are bigger and caudate with large single nuclei. In the neighbourhood of the ciliary body the vitreous is fibrillated in a direction parallel to its surface.

*Optic nerve:* here there is a great increase of round and oval nuclear bodies, none, however, are multi-nuclear.

*Choroid* contains a slight general increase of nuclei, but no multi-nucleated bodies.

*Sclerotic* shows a fairly marked nuclear increase.

Blood-vessels of eye generally contain slight increase of their white blood-corpuscles which are multi-nuclear.

No. 246. John W——, æt. 50, under care of Mr. Wordsworth. *Left eye.* Wound of lower part of cornea 32 days ago. Iris adherent to scar of wound; extensive posterior synechiæ.

Tension diminished. Arteries of ciliary body not enlarged.

Retina detached, except from its anterior and posterior attachments and semi-contracted. Remains of vitreous greenish-yellow and translucent above, whitish-yellow and opaque below near wound. Microscopically it consists of cells, most of which (diam. about ·020 mm.) are exactly like vitreous corpuscles; some others are a little smaller, with 2, 3, or 4 nuclei. Towards the periphery of the vitreous the larger are less abundant, and the smaller lie in the meshes of a reticulum produced by the

coagulation of some albuminous material. Many cells similar to these last lie among the vastly elongated columnar elements of the pars ciliaris retinæ. Its pigment epithelium layer is much disturbed; its pigment granules being scattered and raised from their basement membrane by the interposition of many unpigmented nuclear bodies of moderate size.

Ciliary body; muscular fibres not inflamed, though there is some excess of nuclei in the connective tissue between them and in that of its internal part, even close up to the basement membrane limiting it. They are most numerous in the posterior part of the ciliary body. Some fine blood-vessels of new formation enter the vitreous cavity from the ciliary body. They are crammed with multi-nucleated colourless corpuscles exactly like the smaller ones of the adjacent vitreous. In the blood-vessels of the more distant parts of the eye, *e.g.*, the choroid and optic nerve, the colourless corpuscles, though multi-nucleated, are little in excess.

The patient was anæmic in appearance. He died a year later, apparently of some chest affection.

No. 469. Adelaide H——, æt. 17 months, under care of Mr. Tweedy. Injury 5 days from a penetrating wound of the inferior part of cornea.

*Now:* T. 1.; cornea dull: aqueous purulent: prolapse of iris, iritis: vitreous is a small yellowish globe behind lens. Retina *in situ.* O.D. much swollen. Exudation on ciliary processes.

*Microscopical appearances: P.C.R.* The columnar elements internal to pigment layer are vastly elongated: apparently several nuclei in each at different levels but all these possess the same quite distinctive character, *i.e.*, are large, oval, and pale.

The *pigment* of its epithelial layer is brown and scattered, but not apparently from increase of pigment cells as such, but from dissemination of their brown pigment granules intermixed with which are numerous very deeply staining large oval nuclei, but which cannot be well defined.

There is a great increase of elongated nuclei in the fibres of the ciliary muscle, and many in the connective tissue between them and on their inner surface, but no multi-nuclear bodies anywhere. There are remains of large vitreous cells at back of lens capsule (diameter ·022 mm.): outline indistinct; some with

faint nuclei others more distinct; intermixed with these are other smaller multi-nucleated cells, *i.e.*, with 3 or 4 nuclei clustered together.

*O.D.* swollen: on it a mass of vitreous corpuscles (diameter ·02 mm. after hardening); their nuclei usually lateral and faintly staining; some with no nucleus. Some smaller ones with nuclei dividing as in front part of vitreous.

Walls of blood-vessels in papilla extremely thick: nuclei increased in their outer part and in the loose tissue around them. The white blood-corpuscles within arteries of optic nerve are multi-nuclear. Excess of nuclei in connective tissue of nerve, but none show any evidences of proliferation.

The *corneal* corpuscles are large and irregular both in shape and distribution.

# ON THE PHENOMENA OF SUPPURATIVE HYALITIS AND THEIR RELATION TO THE "MIGRATION THEORY."

By R. G. Hebb, M.D., and W. A. Brailey, M.D.

These observations are made upon cases in which suppuration has occurred either spontaneously in a defective eye or in direct consequence of some local injury. The first stage of suppuration is that the vitreous becomes turbid with fine opacities, and rather more consistent. Retaining strongly its front attachments it separates readily from the retina behind, though less easily from the papilla than elsewhere.

As the suppurative process goes on, its anterior and central part becomes greenish-white and opaque, and the ciliary body is of a similar colour. A comparatively clear zone continuous behind with the similar posterior part of the vitreous, separates the two. The swollen papilla is also covered with opaque substance, from which white streaks radiate into the adjacent vitreous. The vitreous where it is in contact with the retinal arteries is also streaked with faint white lines corresponding to them. These lines broaden until the retina becomes covered with a thin opaque layer. The central and marginal opacities encroach upon the intervening clear substance until the entire vitreous has become opaque. The retina is swollen, opaque, and contains many purplish extravasations of blood. Its inner surface is wrinkled. The vitreous becomes semi-fluid and pus-like (its central part first), and finally may disappear altogether, the hyaloid only remaining as a vertical diaphragm extending across the anterior segment of the globe.

But, if the changes be less acute, the remains of the vitreous show some tendency to organization, and form a yellowish translucent well-defined semi-globular mass adhering to the lens-capsule and ciliary body.

The choroid, though thickened and congested, is to the naked eye otherwise unaffected; no purulent exudation is seen on either of its surfaces.

The sclerotic is thickened and succulent; there is much infiltration of albuminous substance into the episcleral tissue.

The iris is more or less inflamed and may be in some cases infiltrated with pus, in which case there is also pus in the anterior chamber.

The microscope shows the central opaque part of the vitreous to be made up of large closely-packed bodies, between which is, in some cases, a scanty, structureless, or finely granular matrix. These bodies almost exactly resemble vitreous cells in their size* (diam = ·024 mm.), in the shape and lateral position of their nucleus (diam = ·0085 mm.). They are fainter, however, in outline, their nucleus is large and fainter, and their protoplasm less clear. They are also infinitely more numerous.

Intermixed with them are some (perhaps 2 per cent.) small cells (diam ·009 mm.), each containing several deeply staining nuclei clustered together. Transitional forms connect these two varieties with each other. A very few solitary rounded or angular moderately staining nuclei with little or no surrounding protoplasm can also be made out.

The translucent peripheral stratum lying on the ciliary body consists of a loose reticulum, the irregular fibrillation of which is clearly due to the coagulation of some albuminous substance in the meshes of which cells similar to those described above are found.

Here the smaller multi-nucleated cells are more distinctly seen and are at least twice as numerous in a given area as in the vitreous centre. The larger cells, though occasionally equal to them numerically, are sometimes scarcely recognisable. A few cells like them, but caudate, may be found also.

The purulent appearance of the internal aspect of the

* All measurements are taken after hardening in Müller's fluid and alcohol.

ciliary body is due to cells just like the smaller multi-nucleated ones already described, distributed sparsely in the sub-hyaloid space but abundantly between the much hypertrophied columnar elements of the pars ciliaris retinæ and also among the somewhat scattered granules of its pigment epithelium. The semi-transparent posterior part of the vitreous is in structure similar to the translucent zone. The opacities in connection with the papilla and in relation with the retinal blood-vessels are due to cells exactly like those found in the more central parts of the vitreous, though on the average they are a little smaller and more definite as to their outline and nucleus.

In those cases where the shrinking vitreous tends rather to partial organization than to entire conversion into semi-fluid pus, blood-vessels enter it from the ciliary body.

Spindle-shaped cells radiating from the ciliary body towards its centre are seen intermixed with the other elements of the vitreous already described. A few newly formed blood-vessels are seen entering it from the ciliary body which are often densely packed with multi-nucleated white blood-corpuscles.*

The ciliary body itself, though containing an excess of elongated nuclei both on and to a less degree between its muscular fibres, is usually free from multi-nucleated cells. In some, however, a very few are present, but they are not distributed more abundantly in the neighbourhood of the basement membrane limiting the pars ciliaris retinæ but rather the contrary, nor have they any special relation to its blood-vessels. Indeed, though found so freely in the adjacent pigment layer and pars ciliaris generally, they cease abruptly at the basement membrane separating it from the connective tissue of the ciliary body proper. And the staining bodies found in the blood-vessels are restricted to their

* Most of the white blood-corpuscles seen in preparations of healthy human eyes, equally fresh and similarly prepared, have also multiple nuclei. It is fair, however, to state that nearly all of these eyes were slightly congested, having been excised on account of tumours in their immediate vicinity.

lumen, and are clearly distinguishable both by their shape and the multiple character of their nuclei from the elongated nuclear bodies of their walls and of the connective tissue around them.

The iris contains an excess of nuclear bodies, elongated or rounded, some of the latter being multi-nucleated. A reticulum which contains a few multi-nucleated cells constituting a hypopyon, is sometimes found in front of the iris. The *choroid* contains an excess of elongated nuclei and, in rare cases, a few multi-nucleated cells. A few of these latter are also (very rarely) visible in the inner layers of the retina. The walls of the retinal blood-vessels are much thickened with excess of elongated nuclei. The optic nerve generally and the blood-vessels of the papilla are in a similar condition.

The sclerotic contains an excess of nuclei.

The corneal corpuscles are enlarged and disposed at various angles. At a later stage they are distinctly constricted or beaded as if dividing. No other corpuscular elements are visible, most certainly none like the multi-nucleated colourless corpuscles within the blood-vessels. The blood-vessels of the eye generally contain a varying excess of colourless blood-corpuscles which are approximately the same size as the multi-nucleated bodies already described as existing in the pars ciliaris retinæ, and like them contain 2, 3, or 4 nuclei aggregated in a cluster. In one case (No. 246) the newly-formed fine blood-vessels in relation to the *shrinking* vitreous are closely packed with them.

The thickened arterial walls contain an excess of elongated nuclei showing no evidences of division. Similar bodies are abundantly found in the connective tissue immediately surrounding them.

The large cells found in the suppurating vitreous are so exactly like normal vitreous corpuscles in size and shape that it is impossible to resist the conclusion that they are derivatives of them by enormous cell multiplication.

There is room for some difference of opinion as to the

origin of the smaller cells. Their multiple deeply-staining nuclei and the forms transitional between them and the larger cells, might indicate that they are these in a state of rapid multiplication. But, on the other hand, their resemblance to the cells found in the pars ciliaris retinæ and to those within the blood-vessels, might suggest that they are importations either from the blood-vessels or from the pigment epithelium layer of the pars ciliaris retinæ.

In favour of the latter is their abrupt limitation to the inner side of its basement membrane and the fact that although diminishing suddenly at the hyaloid, similar cells can be traced as far as the vitreous centre.

With regard to the question of their origin from the colourless corpuscles of the blood, their shape and size would rather favour it, though their peripheral unstaining zone is firmer and more abundant. Against it is their distribution, that is, they are not found more abundantly in the connective tissue immediately outside the capillaries than elsewhere. Indeed, in the majority of cases, their presence is exactly limited to within the lumen of the capillaries. Thus, in eyes which have undergone suppurative hyalitis, two forms of perverted nutrition may be observed. One in which there is an increase in the growth of the individual elements, as for example, the great elongation of the columnar elements of the pars ciliaris retinæ and of the Müllerian fibres of the adjacent retina (individual hypertrophy); another in which there is an increase of their number, as, for example, in the cells of the vitreous, in the nuclei of the muscular fibres and connective tissue of the ciliary body and of the arterial walls, and in certain cells of the pigment-bearing epithelium layer of the pars ciliaris retinæ* (numerical hypertrophy). It appears to us that the colourless corpuscles of the blood

* The loculi formed by its basement membrane contains spheroidal masses of cells, the outermost being pigmented; the more central are those which undergo proliferation. The resulting cells make their escape by rupture of the internal wall of the loculus and then lie among the columnar elements of the pars ciliaris retinæ.

partake of this last mode of increased nutrition while in the blood-vessels in the neighbourhood of the suppuration, and such a view might explain the leukhæmic condition of a person the subject of extensive or prolonged suppuration.

From the above facts we deduce the general conclusion that the migration of the colourless blood-corpuscles bears no part in these cases of suppuration of the vitreous body.

They are thus clearly marked off in their pathology from metastatic suppurations of the eye-ball, where the vitreous cavity has become full of pus from a migration of colourless corpuscles from the blood-vessels.

## ON THE RELATION OF TUMOURS OF THE EYE TO INTRA-OCULAR TENSION.

By W. A. Brailey, M.D., Curator of the Museum.

It has long been a matter of observation that intra-ocular tumours, whether of retina or of choroid, may be a cause of increased tension, though many points of relation between the two conditions are not known or defined.

This paper seeks to show from the evidence of the cases recited in the preceding Report:—

1. That with intra-ocular tumours a stage of very slightly diminished tension precedes the glaucoma; this last being ultimately in its turn followed by a very considerable softening of the eye.

2. That in cases of tumour implicating solely the exterior of the eye, or even the optic nerve sheath alone, glaucoma may be developed as a consequence, though there is no preliminary stage of slightly diminished tension.

3. That the pathological changes accompanying increased tension, whether from intra- or extra-ocular tumours, are those typical of true primary glaucoma.

4. That in every case of tumour, whether intra-ocular of the retina or choroid, or extra-ocular of the conjunctiva, sub-conjunctival tissues, optic nerve, or orbital walls, with normal or even slightly diminished tension, these changes can be observed in process of development.

5. That the course of the pathological changes in a glaucoma may be inferred by a comparison of those observed in cases of tumours in the different stages of tension above referred to.

1. The first point is established by a reference to the cases of sarcoma of the uveal tract, cited in the Curator's Report (Nos. 580, 293, 313, page 232, *et infra.*), and by two similar cases which have very recently occurred at Moorfields.

The diminished tension is so uniform in its presence and degree, that we are warranted in supposing it to be of universal occurrence.

The pathological changes of this stage will be described subsequently. They consist, however, in brief, of a very slight inflammation of the ciliary body and iris, a slight neuritis, generally some little enlargement of the ciliary folds, with a corresponding slight advance of the iris-periphery as a result.

The cause of the diminished tension is, as far as I am concerned, a matter of speculation; perhaps the vitreous shrinks more rapidly than the tumour grows, or its more gradual atrophy may disturb the balance of fluids in the eye by diminished osmosis from the blood-vessels; or, perhaps with more probability, the loss of secreting uveal surface due to its invasion by the tumour may produce an effect greater than any slightly increased secretion in connexion with the inflammation observed at this stage can account for.

I have been able to draw no evidence in support of this, my first point, from retinal glioma, perhaps because, in this disease, the friends of the patient rarely suspect anything wrong previous to the establishment of the tension.

2. With regard to the *second* point, glaucoma is of much more rare occurrence with extra-ocular tumours.

This may be because the tumour is separated by the thickness of the sclerotic from the tissues most implicated in the glaucomatous changes. But perhaps the cause may rather be that these tumours are earlier observed and sooner operated upon.

In one case, however (No. 570, page 230), there was an increase of tension, and it is interesting to note that, though it must have been of extremely short duration, the pathological changes are markedly those found in primary glaucoma.

The cases of extra-ocular tumour have at their earliest stage always normal and never diminished tension.

3. In the case of extra-ocular tumour just referred to as

accompanied by glaucoma, the ciliary body was inflamed and atrophied; the iris was inflamed and its extreme periphery was applied to the cornea. The optic nerve was inflamed and the papilla swollen. The arteries of the ciliary body were dilated and the ciliary folds were a little enlarged.

And in the glaucoma caused by an intra-ocular sarcoma the changes are much the same.

There is, however, in these cases more evidence of inflammation, both in the adhesion of the ciliary folds to the peripheral part of the lens capsule, and in the presence of posterior synechiæ, than is usually the case in primary glaucoma. Organized fibrinous layers on the inner surface of the ciliary body are also more common in tumours. The adhesion to the lens would seem to show that the ciliary folds have been somewhat swollen. The arteries of the ciliary body are enlarged, though in rare cases where the ciliary region is staphylomatous, as, for example, in No. 419, they are not recognizable. Under similar circumstances they may not be noticeable in cases of primary glaucoma.

The optic nerve is much inflamed if the disease has been of short duration; but if it has existed longer, there is great connective-tissue increase of its trabeculæ, especially round the blood-vessels, which are numerous and large. The existence of these changes in the nerve is quite independent of the size or position of the tumour. The changes are greater as the tumour becomes larger. The swelling of the papilla seems always to exist at an early stage of the glaucoma, though it is often but slight. It results in atrophy. I have reason to believe that primary glaucoma is similarly always preceded by hazy and slightly swollen papilla.

In the glaucoma consequent upon retinal glioma the same changes are developed. There is, however, no inflammatory adhesion of the ciliary folds to the lens-capsule, but, on the contrary, the two structures are often separated by a broad stratum of glioma cells. This may perhaps be because the lens is more spherical at the time of life when glioma is more apt to occur.

The optic nerve, though its nerve fibres may be entirely replaced by glioma cells, has its fibrous trabeculæ, as well as its inner and outer sheaths very strongly hypertrophied.

Thus both intra- and extra-ocular tumours are accompanied by the pathological changes characteristic of glaucoma, though, as would be expected, they are most pronounced in the first class of cases.

4. Evidence that these changes are in existence before the tension becomes increased may be found by consulting the cases in the Curator's Report (Nos. 313, 388, etc.). It is difficult to say precisely why there should be inflammation, and still more difficult to say why more distant parts should share in it, for example, the ciliary body in tumour of the optic nerve sheath (No. 570), and conversely why there should be neuritis in tumour of the choroid, or of the external ciliary region (Nos. 548, etc.). It is even more inexplicable why the more remote structure should be sometimes the most inflamed. (No. 548.)

5. Clearly, if these tumours result in tension, and if their pathological conditions are the same as those found in glaucoma, their earliest pathological state is that of the pre-tension stage of glaucoma.

I have previously indicated elsewhere that, in my opinion, such a stage exists, and, among others, such clinical phenomena as failing accommodation, obscurations, enlarged ciliary vessels, and, I believe in every case, haze of the optic disc indicative of optic neuritis point in this direction.

The course of a primary glaucoma would then be, inflammation of ciliary body with enlargement of its arteries and a less degree of inflammation of the iris; enlargement of the ciliary folds with a corresponding advance of the peripheral part of the iris, and shallowing of the anterior chamber. In addition there is optic neuritis with a slight haze and swelling of the papilla.

All these changes are at first extremely slight, but afterwards they increase. The somewhat advanced periphery of the iris is carried forward against the cornea; it becomes

adherent there and finally atrophies. The ciliary folds may become adherent to the lens capsule. The ligamentum pectinatum, at first slightly inflamed, afterwards shrinks up and draws to a point the origin of the radial fibres of the ciliary muscle.

Shrinking, with connective tissue growth, takes place in the ciliary body generally, and its muscular fibres become especially atrophied. The ciliary folds participate in this atrophy, and the space around the lens-margin becomes much more widely opened than normally. They are always widely removed from the iris, from which it might be inferred that, though undoubtedly instrumental in first advancing it, they have not remained long in contact with it. Similar changes occur in the optic nerve. There is a shrinking of its bulk from atrophy of its nerve fibres, and a vast increase in its connective-tissue trabeculæ. These contain blood-vessels beyond the normal both in number and in size. When, however, the nerve is much contracted and hardened, the blood-vessels are less conspicuous.

The sclerotic, at first slightly inflamed and softened, becomes at last somewhat more rigid than normal.

It is worth noting that connective tissue growth is much less pronounced as a stage in the glaucoma secondary to tumours than in primary glaucoma, perhaps because the average age of patients who are subjects of the disease is less. But still an imperfect connective tissue is developed abundantly in the optic nerve in many cases of glioma.

In the cases of tumour where the tension has finally fallen below the normal, the inflammatory have given place to atrophic changes. The arterial enlargement is not found. The pigment epithelial layer on the internal surface of the ciliary body is atrophied, and its pigment granules are scattered.

In connexion with the preceding remarks, certain questions suggest themselves.

A. How does the presence of a tumour produce inflammation, especially that of the ciliary body and the optic nerve ?

An increase of nuclei is always found in the tissues bordering on a tumour, for example, with epithelioma of the conjunctiva (No. 56) it exists in the loose sub-conjunctival tissue. And sarcoma of the optic nerve-sheath produces inflammation and connective tissue growth in the nerve. Choroidal sarcoma may be a cause of iritis and resulting posterior synechiæ. And glioma of the retina produces similar changes. These inflammatory conditions are not due to commencing tumour extension, but rather to the physical influence of the tumour, for a cyst pressing upon the optic nerve produces precisely similar changes.

B. Supposing that a tumour can inflame the tissues immediately in relation with it, how is it that the inflammation travels to more distant parts, for example, from an internal or external tumour of the ciliary region to the optic nerve, or from tumour of the optic nerve-sheath (No. 570) to the ciliary body, or from the retina to the optic nerve and ciliary body ?

Microscopical examinations show clearly that neither the vitreous body nor the retina is the carrier of inflammation, but that the fibrous and vascular tissues of the eye are the parts concerned in it. Thus in (No. 570) inflammation can be clearly traced forwards along the episcleral web and to a less degree along the choroid. More commonly, however, the last appears to take no share in it.

A similar condition of things occurs with wounds or ulcers of the cornea. The optic neuritis that is universally found under these circumstances appears to have been transmitted backwards from the cornea by way of the sclera and episcleral tissue.

C. How do the inflammatory and vascular changes described above stand in relation to the tension ?

We must imagine that a hyper-secretion due to the inflammation of the ciliary body can produce the tension, though the earliest stages of inflammation co-exist with a tension even below the normal. It is, of course, impossible that atrophic changes can of themselves produce increase of fluid. It is

important to remark the frequency of arterial enlargement in the ciliary body.

D. Are there any other physical causes in action to which we can attribute the increase of tension ?

Undoubtedly the application of the peripheral part of the iris to the cornea is a strong factor in preventing the escape of fluids from the eye. And it exists in every case of glaucoma secondary to tumour contained in the previous Report.

There is no evidence in any of the cases of any obstruction to the passage of fluids round the lens-margin as a cause of the glaucoma. Indeed, in the cases where the ciliary body is vastly thickened by the tumour (Nos. 375 and 313) the tension is actually below the normal.

E. What is the cause of the advance of the peripheral part of the iris ?

In choroidal tumours some advance is due undoubtedly to the enlargement of the ciliary folds, which are extended equally in all directions. But only one case (No. 121) gives evidence that they have actually pushed the iris against the cornea. Indeed, it is the rarest thing in the world for the iris periphery to adhere to the ciliary folds, as it would probably have done had the two remained long or been forcibly in contact, whereas adhesion of the lens-capsule to ciliary folds is not uncommon.

It seems that an advance due, in the first place, to the ciliary folds, has been made complete and permanent by pressure of fluid, accumulated from obstruction to its exit from the eye in its passage along the shallow anterior chamber in its course to Schlemm's canal.

Though the ligamentum pectinatum becomes much contracted, it is clear that in eye-tumours this change does not occur early enough to retard by its effect upon the iris the exit of fluids from the eye.

In retinal glioma the ciliary folds are often clearly seen advanced to the iris. This, however, appears to depend in many cases upon the actual filling of the vitreous chamber by the tumour.

## ON THE NATURE AND COURSE OF THE GLAUCOMATOUS PROCESS.

By W. A. Brailey, M.D.

A sufficient number of eyes at various stages of glaucoma have now been examined to warrant us in speakign on this disease with precision.

It is certain that inflammation of the ciliary body, iris, and optic nerve is always present, and that it is one of the earliest symptoms of primary glaucoma, being developed previous to the tension. It is usually most pronounced in the ciliary body, especially in and around its muscular fibres.

The iris periphery is nearly always applied to the cornea, and its advanced position is caused in the first instance by an enlargement of the ciliary folds due to their vascular turgescence, but afterwards its application is rendered more close and more extensive by the pressure of fluid behind it. Coincidently with the enlargement of the ciliary folds the arteries of the ciliary body become dilated.

Very shortly the ciliary body becomes altered in shape by the stretching of its muscular fibres in a meridional direction: this and their atrophy render its thickness somewhat less. Its arteries remain dilated though the ciliary folds are emptied of blood, and small. These and the antero-internal part of the ciliary body and ciliary muscle recede backwards so as to be widely removed from the iris periphery.

Subsequently there arises very rapid atrophy of the ciliary muscle fibres with very commonly dense connective tissue formation between them and in the optic nerve.

The choroid is compressed and atrophic. It rarely presents any traces of inflammation. The lens has not exceeded the normal diameter in any one of the specimens examined, and in most it has been considerably flattened in an antero-posterior direction.* It has been advanced but very rarely: the posterior chamber of the aqueous is usually of great depth.

* *See*, however, Vol. x, Part I, p. 131, Fig. 16.

If we ask for the causes of the increased intra-ocular fluid we must attribute it in the first instance to an inflammatory hyper-secretion of fluid from the ciliary body and iris. But when the iris has once become applied to the cornea another cause is established in an obstructed outflow of fluid from the globe through Schlemm's canal. When atrophic changes have supervened in the ciliary body and iris, this occlusion of the entrance to Schlemm's canal remains as the sole agent in keeping up the tension. Iridectomy cures by removing the periphery of the iris, and is effective provided that the inflammation is not in an acute stage.

But in glaucoma of long standing the atrophied iris is so firmly adherent to the cornea that its removal by iridectomy cannot be effected.

In such cases as well as in others sclerotomy may cure by making a new access to Schlemm's canal.

"Hyposcleral Cyclotomy" may cure in a similar manner, but it will be less effective than sclerotomy, as it makes a new access at one point only.

The glaucomatous inflammation is characterized for the most part by an absence at any stage of any thickening of the ciliary body and iris, and by the lack of fibrinous exudations on them. As compared with other forms of inflammation, it shows only a moderate increase of nuclei in the parts it involves. In some cases there is evidence that the arterial dilatation is secondary to an obstruction in the circulation caused by increased intra-ocular pressure. But it remains remarkable that this enlargement is not seen in the increased tension accompanying serous inflammations of the uveal tract.

The changes which are found to occur in Primary Glaucoma are in nearly all respects the same as those inferred from the preceding study of the increase of tension accompanying Tumours.

## ON THE MEANING OF THE WORDS "NYCTALOPIA" AND "HEMERALOPIA:" WITH A CRITICAL EXAMINATION OF THE USE OF THESE WORDS IN THE ANCIENT GREEK AND LATIN AUTHORS.

By W. A. Greenhill, M.D., Oxon.

[Notwithstanding what may be almost called a general consensus of opinion among modern writers regarding the meanings of the words nyctalopia and hemeralopia, I have from my first examination of the subject, some nine or ten years ago, thought that, according to the authority of the best classical writers, and according to the etymological construction of the word, the real meaning of the term nyctalopia is *night-blindness*, and not *night-sight* as the moderns assert; and consequently, that the meaning of the opposite term hemeralopia is *day-blindness*, and not *night-blindness*. A further special investigation two years ago confirmed me in this opinion. Failing, however, to obtain the acquiescence of those of my friends who were acknowledged authorities in all matters relating to classical learning, I determined to submit the question to Dr. Greenhill, who has given much attention to ancient medical nomenclature. Accordingly, about fifteen months ago, without stating the conclusion to which I had come, I asked Dr. Greenhill to be so good as to favour me with his opinion on the true classical meaning of the word nyctalopia: and was not a little gratified to find that he had, from a more complete and critical examination of the authorities than I could pretend to have made, come to the same conclusion as that at which I had arrived. Dr. Greenhill has kindly furnished me with an elaborate statement of his views in a letter which, with his permission, I have much pleasure in publishing. The Editor.]

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Dear Sir,—I quite agree with you in thinking it most desirable to endeavour to fix the meaning of the words "Nyctalopia" and "Hemeralopia;" and, as you ask for my opinion on the matter, I will give it in some detail, and will state the reasons for thinking that it will be better for the future to use the word

"Nyctalopia" in the sense of night-*blindness*, rather than night-*sight*.

When a person invents a new name, whether it is formed with etymological accuracy or not, he has a right to fix to a certain extent its meaning, which all who make use of his word will refrain from altering without some sort of necessity. But in the case of "Nyctalopia" and "Hemeralopia" we do not know who invented the words, nor what they were originally intended to signify, except that evidently one was meant to be the opposite of the other.

In most cases, however, (except in some of the barbarous and unintelligible terms of the present day,) the meaning of a compound-name is indicated with sufficient clearness by its etymology. But here, again, we are at fault in the case of the terms in question, for (in the opinion of the best etymologists) the force of the different syllables and *letters* of which the words are composed is by no means incontestably ascertained.

Perhaps, therefore, upon the whole it is safest to try to ascertain in what sense the words were used by the earliest Greek and Latin writers, even though there is not an absolute *consensus* of authorities, and one Greek glossographer candidly gives *both* meanings.[1] Nevertheless, it will appear that the preponderance of the ancient authorities is decidedly in favour of the meaning given above, and this is probably the strongest reason for adopting it.

Before beginning to examine these ancient writers, it may be well to state that the following remarks will apply exclusively to "*Nyctalopia*," the corresponding word "*Hemeralopia*" being probably found only *once* in any ancient Greek or Latin writer, and it being impossible to decide the sense in which it is used in that passage.[2]

In conducting such an inquiry, the first step would naturally be to find out the meaning given to the word by Hippocrates, Aristotle, and Galen, and their usage of the term might almost be considered as final and conclusive. The case of Hippocrates

[1] The author of the spurious work called "Introductio, seu Medicus," cap. 16, in Galen's works, tom. xiv, p. 776, l. 9.

[2] Pseudo-Gal., "Introd.," p. 768, l. 8. The word has escaped the notice of the Paris Editors of H. Stephani "Thes. Græc.," and also of Liddell and Scott.

must be especially noticed presently, and Aristotle does not define the word;[1] but Galen says distinctly that it was intended to signify night-blindness,[2] and that this is the sense in which it is used in the Hippocratic Collection:[3]—it is probable also (though it cannot be positively proved) that this is the meaning which he gives to the word himself.[4]

The evidence of Pliny is valuable, as showing that the word was not in his time thoroughly naturalized among the Romans, but was only beginning to be used;[5] while Celsus, who lived somewhat earlier in the same century, mentions the disease, but without giving it any name.[6] How then can Celsus be quoted as an authority for the meaning of a word which he does not use? The answer is perfectly satisfactory; it is because he is almost certainly referring to a passage in the Hippocratic Collection, which will be mentioned presently; and if your oppo-

[1] "De Gener. Animal.," v. 1, § 28, p. 146, ll. 15, 19, ed. Bekker.

[2] "De Meth. Med.," ii, 2, tom. x, p. 84, ll. 11, 13, 14.

[3] "Gloss. Hippocr.," tom. xix, p. 124.

[4] See "De Simplic. Medicam. Temper. ac Facult.," i, 1, § 11; "De Compos. Medicam. sec. Loc.," iv, 8, tom. xii, p. 336, l. 3; p. 802, l. 10. At first sight these passages would seem to prove nothing, as no explanation of the word is given; but the mention of "goat's liver" as a remedy for the disease, seems to point out that it is *night*-blindness that is intended. The explanation of this empirical mode of treatment is to be found in two passages of Pliny ("Hist. Nat.," viii, 76, p. 100, l. 9, ed. Tauchn; xxviii, 47, p. 192, l. 4 from bottom), in which he says that goats can see as well by night as by day, and that therefore their blood and liver are used in cases of *night*-blindness. Of course this argument is not logically conclusive, because we are not sure that goat's blood and liver may not have been given in cases of *day*-blindness also; but it seems to amount to a high degree of probability, and may be used (*valeat quantum valet*) until it is shown to be fallacious.

This same argument applies to Dioscorides, who says that goat's liver is useful in cases of *nyctalopia* (*i.e.*, *night*-blindness?) "De Mat. Med.," ii, 47, tom. i, p. 185, l. 7; ed. Spreng.

Two apparent exceptions to the statement in the text ("Introd.," cap. 16, and "Defin. Med.") will be noticed presently; and two other passages in the Galenic Collection *in accordance with* those here referred to, need not be added to the list, because the work in which they occur is (probably) spurious. ("De Remed. Parab.," i, 5; ii, 4, tom. xiv, pp. 350, 415.)

[5] In one passage ("Hist. Nat.," xxviii, 47), he speaks of "Nyctalopas *a Græcis dictos*;" in two others (viii, 76; xxxii, 24) he uses the name like an ordinary Latin term.

[6] "De Medic.," vi, 6, § 38.

nents are inclined to dispute this, you may fairly ask what other word they think Celsus had in his mind.

The succeeding writers who use "Nyctalopia" in the sense of *night*-blindness may be simply enumerated chronologically, viz.: Oribasius,[1] Palladius,[2] Aëtius[3] Alexander Trallianus,[4] Paulus Ægineta,[5] and Joannes Actuarius.[6] A few more authorities might be added, but you will be inclined to cry out, "Ohe, jam satis est."

It is indeed a goodly list, containing the names of all the most eminent physicians of antiquity, with *one* exception, viz., *Hippocrates;* and as long as your opponents are able to appeal to Hippocrates and Galen in favour of their interpretation of the word, there is no prospect of the discussion coming to a satisfactory termination. It is therefore necessary to examine the passages in these authors where the word occurs; and we will begin with Galen.

It has been already stated that in one of his works, which is undoubtedly genuine, Galen distinctly explains Nyctalopia to signify *night*-blindness, and it is almost incredible that so careful a writer should be found to use the word elsewhere in the opposite sense. The two passages alleged in support of this inconsistency are found in two works, which five-and-thirty years ago I stated (after Ackermann, Kühn, and Choulant) to be of doubtful genuineness.[7] I know of no reason for modifying the opinion which I then expressed, but should

[1] "Synops.," viii, 48; "Eupor. ad Eunap.," iv, 18, tome v, pp. 451, 709, ed. Daremberg.

[2] Dietz, "Schol. in Hippocr. et Gal.," tom. ii, p. 181, ll. 12, 18, 24. This passage is curious from the mention of ἀλώπηξ, *fox*, in connection with the disease, which perhaps accounts for the monstrous word νυκταλώπηξ, found in some editions of Aristotle (*loco cit.*), where Bekker properly reads νυκτάλωψ.

[3] VII, 46, p. 132, l. 50, ed. Ald.

[4] II, 6, p. 147, ed. Basil.

[5] III, 22, p. 35B. l. 23, ed. Ald. (See vol. i, pp. 420, 434, Adams's Transl.)

[6] "De Diagn.," ii, 7, p. 447, l. 32, in vol. ii of Ideler's "Phys. et Med. Græci Min."

[7] The "Introductio, seu Medicus," c. 16, tome xiv, p. 776, l. 9; and the "Definitiones Medicæ," c. 343, tom. xix, p. 435. See Smith's "Dict. of Gr. and Rom. Biography," vol. ii, pp. 216, 217.

rather be inclined to intensify it, and to argue that the fact of their contradicting in this point Galen's known opinion, is a plain proof of their spurious origin. Unless, therefore, your opponents are prepared to support the genuineness of these two works, they cannot appeal to the authority of Galen.

The case of Hippocrates requires more discussion, but the details shall be relegated to a *Note*, so that they need not be read by any one who does not care to enter upon a minute point of verbal criticism. The word occurs in *five* passages in the Hippocratic Collection, but of these only the third concerns us here, as the other four merely mention the term, without any indication of its meaning.[1] This passage requires very different treatment from those (supposed to be) quoted from Galen, which have just now been noticed; but the result will be equally satisfactory. In this case it is not sufficient to state that it comes from one of the spurious treatises, because its being found in any part of the Hippocratic Collection gives it a priority of several centuries in antiquity over the earliest passage quoted on our side. Indeed, until the year 1864, your opponents had a perfect right to appeal to the Hippocratic Collection, for up to that date the reading in every (?) edition was οἱ δὲ τῆς νυκτὸς ὁρῶντες, and Ermerins was the first editor, who, by introducing the word οὐχ before ὁρῶντες, completely altered the sense.[2] I believe myself that he was right in making this change,[3] and unless your opponents can show with some degree of probability that he was wrong, they must not appeal to Hippocrates for the future.

It seems almost ludicrous to mention in opposition to the authority of Oribasius, Aëtius, Alexander Trallianus, and Paulus

[1] "Epid.," iv, § 52; vi, 7, § 1, tome v, p. 192; p. 332, ll. 2, 3, 5, ed Littré. "Prorrhet.," ii, 33, 34; "De Visu," § 7, tome ix, pp. 64, 66, 158.

[2] As his edition is by no means convenient for use, it may save trouble if I mention that the passage will be found in vol. iii, p. 389.

[3] My reasons for this opinion are *four*, viz: 1, there is sufficient MS. authority for the reading; 2, it was probably so written in the MS. used by Celsus, and 3, also by Galen; and 4, *Nyctalopia* has not been found in the sense of *day*-blindness in any author so early (by many centuries) as the Hippocratic Collection. These four points (or at least the first three) require some explanation.—

1. The learned Greek, Coray, at the beginning of this century, pointed out the fact (which had before been noticed by Chamseru in the "Hist. de la Soc. Roy. de Méd.," 1786, Pt. ii, p. 141) that one of the MSS. in the great

Æginetа, such names as Theodorus Priscianus,[1] Leo,[2] &c.; so that the cause of your opponents, so far as it rests on classical authority, may be said to have broken down altogether.

But though the chief reason for wishing to restrict the signification of *Nyctalopia* to *night*-blindness is the use of the word in that sense by the old medical writers, you can also derive some little support from etymology,—at least, as much as your opponents. The derivation of words ending in ωψ is, I believe, very puzzling and uncertain; but here I would speak with great diffidence, as I am aware that I am getting *ultra*

Library at Paris (which has changed its name so many times since then) had originally the word οὐχ before ὁρῶντες, which had been erased, but was still quite visible." ("Notes sur le Traité des Airs," &c., p. 46.) This MS. (No. 2254) is of the 14th century, and is placed by M. Littré *fourth* in order of importance among the general MSS. of Hippocrates (tome i, p. 518; tome ii, p. 378), and *first* among those that contain the "Prorrhetica" (tome ix, p. 4). Nevertheless, M. Littré has not admitted this reading into his text, (1) because it is found in no other MS., and (2) because the ancient writers were not agreed as to the sense of *Nyctalopia*. But surely no one knows better than M. Littré himself that the reading of a number of inferior MSS. may be rejected in favour of a superior one; and, if he had examined the passages in which *Nyctalopia* is used by ancient writers, he would have found such a preponderance in favour of *night*-blindness as would have induced him to correct his text, and thus anticipate Ermerins (Hippocr. "Opera," vol. iii, p. 389).

2. Coray refers to Celsus (vi, 6, § 38), and points out that he must certainly have been translating this passage of the "Prorrhetica." If this be so, it shows us the state of the text at the beginning of the Christian era, twelve hundred years before the date of the earliest existing MS., and is a testimony of the very highest importance.

3. Scarcely less weighty is the fact that Galen, in the latter part of the second century, interpreted *Nyctalopia, as used in the Hippocratic Collection*, to signify *night*-blindness, which is quite a different thing from saying that he used the word in that sense himself. It amounts, therefore, to a moral certainty that Galen (if in those early times there was any diversity in the MSS. which he used) considered the true reading to be οὐχ ὁρῶντες.

4. I believe (but, of course, cannot prove) that *Nyctalopia* did not bear the sense of *day*-blindness when the "Prorrhetica" were written; and this belief may very fairly be held until some early instance of this use of the word is brought forward.

[1] I, 10, quoted by Forcellini (*Facciolati*).

[2] "Consp. Medic.," iii, 39, in Ermerins, "Anecd. Med. Gr.," p. 149.

*crepidam*. Without therefore noticing any of the other words with this termination[1] (many of which have nothing to do with ὤψ), we may be content in the cases of ἡμεράλωψ and νυκτάλωψ to accept Galen's etymology,—at least until any manifestly superior derivation has been brought forward. Of course his authority in matters of etymology is not very great, for etymology was not studied scientifically among the ancients; but we know, that, among all other subjects connected with his profession, he had given much attention to medical nomenclature,[2] and it is probable that he had taken some pains to ascertain what the word was really intended to mean. He explains νυκτάλωπες, *οἱ τῆς νυκτὸς ἀλαοί*,[3] *those who are blind* (ἀλαοὶ) *by night*, evidently supposing the word to be compounded of νύξ ἀλαός, and ὤψ, and thus accounting for the αλ in a simpler manner than any other of the etymologies which have been proposed,[4] and which need not be examined here.

It is quite unnecesary to mention the opinions of modern lexicographers in detail; some explain the words in one way, and some in the other, perhaps without any very decided preponderance on either side. There is, however, one great living authority, whose reasons for preferring the wrong (?) interpretation require to be examined in detail, on account of the great and deserved weight that belongs to every opinion (on such subjects) that he expresses. I confess I feel something like a parricide in thus "laying hands on my father Parmenides;"[5] but, if these pages should ever fall under his notice, I am sure he will excuse my thus disputing his opinion.[6] M. Littré has in *three* places explained *Nyctalopia* by *day*-blindness, viz., in his edition of Nysten's "Dict. de Médecine" (1855); in the Notes to

[1] Viz., ἀλαώψ, μύωψ, ὕδρωψ, μώλωψ, ἀγχίλωψ, αἰγίλωψ, αἱμαλώψ, θυμάλωψ.

[2] See "Meth. Med.," ii, 2, tom. x, p. 81, &c.

[3] "Gloss. Hippocr.," tom. xix, p. 124.

[4] Liddell and Scott derive the word from νύξ and ὤψ only, ignoring the αλ altogether; but as they recognize the compounds ἀλαωπός, and ἀλαώψ, if we prefix νυκτ to these words, we have *very nearly* the word we want.

[5] See Jowett's Plato, Preface.

[6] [Since these words were written, the death of M. Littré has been announced; but the sentence may be left, as a slight token of respectful regret for the loss sustained by his friends and by the literary world. June, 1881.]

his Hippocrates (1861); and in his "Dict. de la Langue Française" (1878).

1. In his "Dict. de Méd." he says that "la lettre *l* est purement euphonique," and derives the word from *νύξ* and *ὄπτομαι*, noticing the derivation from *νύξ*, *ἀλαός*, and *ὤψ*, only to reject it: but as the etymology of the word has been noticed above, perhaps it is sufficient here to say that his derivation is not evidently superior to Galen's. He says that Hippocrates and Galen used the words *νυκτάλωψ* and *νυκταλωπία*[1] in the sense of *day*-blindness: but in the case of Hippocrates he should have stated that the reading of the passage to which he referred was doubtful; and in the case of Galen he should have said that the two treatises in which the word is used in this sense are at least of doubtful authenticity, while in one of Galen's genuine works the word is used in the sense of *night*-blindness.

2. His Note on Hippocrates (tome ix, p. 65) has been already considered.

3. In his "Dict. de la Langue Française" under *Héméralopie*, he quotes the Greek form *ἡμεραλωπία*, which (as has been said before) has not hitherto been found, and calls it "composition irrégulière de *ἡμέρα jour*, et *ὤψ*, *ὠπός*, *œil*." He gives no special reason for preferring the meaning assigned to this word and to *Nyctalopie*, and seems to rely entirely on the etymology, which (it is submitted) cannot be considered sufficient to decide the question.

Under *Nyctalopie* he says, "La forme s'explique par une forme *νυκταλός*, qu'on trouve au sens de *dormeur*," and derives the word *νυκτάλωψ* from *νύξ* and *ὄπτεσθαι*, as in his "Dict. de Méd." But *νυκταλός* seems to be a suspicious word (see Liddell and Scott), and there is no corresponding word, *ἡμεραλός*, to appeal to in the case of *Héméralopie*: so that, upon the whole, while we may be quite inclined to agree with M. Littré in considering the composition of the two words to be anomalous, we may at the same time be willing to acquiesce (at least for the present) in an etymology with which Galen appears to have been satisfied.

This is what I had to say on the subject, but I must not

[1] This is not quite correct, for the form *νυκταλωπία* is not found in Greek; nor is *ἡμεραλωπία*, which M. Littré gives under *Héméralopie*.

end my letter without offering you a few words of encouragement, together with my reason for thinking it possible that you may be successful in your endeavour to put an end to the confusion which has existed for considerably more than a thousand years. Coray hoped "faire cesser une fois pour toutes cette confusion qui regne depuis le siècle de Galien," and he did his part in the work by reproducing M. Chamseru's correction of the passage in the "Prorrhetica." The emendation was not, however, brought sufficiently before the members of his profession, but was buried in the Notes to his edition of the "De Aëre, Aquis, et Locis;" neither was it adopted by the subsequent editors of the Hippocratic Collection. Now, however, that Ermerins has introduced it into his edition,[1] the case is altered, and you have the advantage of being able to refer your opponents to the printed text, where they will find the corrected reading. The other reason why Coray failed in his attempt was because (though he must have known it) he did not point out the worthlessness of the passages quoted from spurious Galenic treatises. Now that this has been done, thus knocking away the *second* mainstay of the building, you may perhaps without unreasonable presumption hope to see the whole edifice fall to the ground.

Yours truly,

W. A. Greenhill, M.D., Oxon.

Hastings, *April* 23, 1881.

Note.—It is a curious coincidence (if nothing more) that the Latin terms *lusciosus*, *luscitiosus*, after being used by Varro[2] and Pliny[3] to signify *night*-blindness, were by later writers[4] used in the sense of *day*-blindness.

[1] 3 vols, 4to. Traj. ad Rhen., 1859–64. It is a great pity that Ermerins should have preferred typographical splendour to utility, and should have published both his Hippocrates and his Aretæus in such a cumbrous and expensive form as to prevent their ever coming into general use. At last, however, he was sensible of his mistake, and his Soranus is equally elegant, and far more convenient.

[2] Quoted in Forcellini (*Facciolati*).

[3] "Hist. Nat.," xxviii, 47, p. 192, l. 4 from bottom, ed. Tauchn.

[4] Pompeius Festus, *in voce;* Fulgentius, quoted in Forcellini (*Facciolati*). The passage in Plautus ("Mil. Glor.," ii, 3, 50) may perhaps be taken in either sense.

In this Letter the common Latin form *Nyctalopia* has been used, but there is probably no such word in Greek, and perhaps the earliest writer who uses it is Isidorus Hispalensis ("Orig." iv, 8, quoted by White and Riddle). The Greek forms are the following: νυκτάλωψ, meaning sometimes the *disease* itself (Hippocr. "Epid.," vi, 7, § 1, tome v, p. 332, ll. 2, 3, 5; Arist. "Gener. Anim.," v, 1, § 28, p. 146, ll. 14, 19), sometimes the *patient* (Hippocr. "Epid.," iv, § 52, tome v, p. 192, l. 4; Pliny, "Nat. Hist." xxviii, 47, p. 192, l. 4 from the bottom); νυκταλωπικός (Hippocr. "Epid.," vi, 7, § 1, tome v, p. 334, ll. 6, 8); νυκταλωπιάω (Gal. "De Comp. Medic. sec. Loc." iv, 8, tom. xii, p. 802, l. 12; Aëtius, vii, 2, p. 124, l. 16); νυκταλωπίασις Orib. ("Eupor.," iv. 18, tome v, p. 710, l. 2).

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Part I.

## Original Contributions.

### ON A CASE OF SPURIOUS NEUROMA OF THE OPTIC NERVE.

By J. W. Hulke, F.R.S., F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital; Surgeon to the Middlesex Hospital.*

Post-ocular intra-orbital tumours of the optic nerve, and of its sheath, are of such rare occurrence that the following case appears worthy of permanent record. The extremely slow rate of increase of the proptosis, indicative of correspondingly slow growth of the tumour, and the relative freedom of the movements of the eyeball, together with the normal appearances of the fundus oculi, had inclined me to regard exostosis as the most probable cause of the proptosis, and to reject as improbable a sarcoma or carcinoma of the orbital walls or optic nerve. This, however, proved erroneous.

Caroline ——, æt. 19, a thoroughly healthy-looking brunette, was admitted on 26th July, 1881, into the Middlesex Hospital with extreme proptosis of the right eyeball, to the extent that even with a strong effort she could not close the eyelids, and sometimes in the attempt they slipped behind the eyeball. This occasionally happened in the act of winking, and it was so painful to her, and the effect so distressing to those about her,

that she shunned society, and was rendered more unhappy by this than by the loss of sight.

The eyeball moved consensually with the other, and nothing unnatural could be felt within the borders of the orbit by pressure through the eyelids. Vision was reduced to quantitative perception of light. With the Ophthalmoscope nothing unnatural was discernible in the optic nerve or retina. The external aspect of the eyeball was perfectly natural.

*History.*—An elder sister, who accompanied her, said that the unnatural prominence of the eyeball began to be noticed in her sixth year, and that its increase had been very gradual, and unattended with pain, but with progressive loss of sight.

The patient was so anxious to be relieved of the disfigurement that she had come to London for the purpose of having the eyeball removed, should this prove necessary.

I decided to examine the back of the orbit through an incision in the conjunctiva at the inner side of the eyeball. The inner border of the orbit was chosen because I had found this more often than the other walls to be the seat of exostosis. When the finger-tip was passed in behind the eyeball it encountered in the bottom of the orbit a tumour so intimately connected with the optic nerve that it could not be separated from it. The eyeball and the tumour were therefore removed together, the optic nerve being severed close to the foramen opticum.

At the end of a fortnight the patient returned to the country wearing an artificial eye, with a corresponding improvement in her personal appearance, which was highly gratifying to her and her friends.

The tumour was firm, of a spherical outline, in size about one-third less than that of the eyeball, from which it was separated by a portion of seemingly healthy optic nerve, somewhat more than half an inch long.

The extremely slow growth of the tumour (dating its beginning from the time when the protrusion of the eyeball was first noticed) is a very remarkable circumstance, and one well calculated to lead to a mistake in diagnosis. Dr. Brailey has kindly furnished the accompanying report of its structure.

The short piece of nerve that intervenes between the eye-ball and the tumour is folded on the latter in such a way that the two bodies lie almost in apposition.

Except for some thickening, the dural sheath passes unchanged over the mass, but the loosely-arranged fibres of the intervaginal space are enormously increased in the neighbourhood of the entrance of the nerve into its front part. They also bear far more nuclei than normal. Towards the centre of the tumour they gradually become replaced by an imperfectly fibrillated tissue containing numerous cell elements, of which the majority are small, perhaps ·005 mm. in diameter, and only slightly elongated, with dot-like nuclei. From them all gradations of size are found up to the largest, which are ·026 mm. in diameter, oblong, and perhaps slightly drawn out at the ends, with several dot-like nuclei and numerous granules. The pial sheath and nerve-fibres lose themselves in approaching the centre of the tumour. The new growth appears to be a sarcoma, taking its rise in the loose tissue of the intervaginal space, most probably from the nucleated cells found upon its fibres.

## ON A CASE OF SARCOMA SPRINGING FROM THE SHEATH OF THE OPTIC NERVE—EXCISION OF THE GLOBE AND REMOVAL OF THE TUMOUR—RECURRENCE—DEATH—NECROPSY—SECONDARY DEPOSITS.

By George Lawson, F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital.*

Tumours of the optic nerve are comparatively rare. They may occur in two ways—1. They may be external to the nerve-fibres, and spring from the dural sheath of the nerve. 2. They may grow from within the nerve, being intimately associated with the connective-tissue between the nerve-fibres, and constituting a neuroma. The following case is an example of a sarcoma springing from the dural sheath of the optic nerve. In the next number of the Reports I will give the case of a large neuroma of the optic nerve, which projected the eye completely beyond the orbit.

Samuel S., æt. 65, was admitted into the Royal London Ophthalmic Hospital on the 26th October, 1880, suffering from all the symptoms of an orbital tumour behind the left eye. The eyeball was bulged considerably forwards and a little outwards, and was immovable. The cornea was clear; there was a good anterior chamber, the pupil dilated and inactive; no perception of light. The eyelids were swollen and closed on the protruding eye; and the conjunctiva was also swollen and chemosed.

*History.*—The patient was unable to give any satisfactory account of the progress of the case. He was apparently very indifferent and apathetic, and had not really detected that anything was wrong with the eye until it began to protrude.

He stated that he first noticed a hardness around the eye about five weeks before his admission into the Hospital, and that this rapidly increased and caused the eye to bulge.

On November 11, 1880, having first excised the globe, I

then removed a solid tumour which occupied the orbit, and completely surrounded the optic nerve.

Dr. Brailey, the Curator of the Museum, made a section of the globe, and a microscopical examination of the tumour.

The parts within the eye were normal, except the optic disc, which was greatly swollen from the pressure of the tumour in the orbit.

The growth was a round-celled sarcoma, and appeared to spring from the sheath of the optic nerve. The drawings show a section of the tumour, and also the manner in which the neoplasic elements had infiltrated themselves amongst the fat vesicles of the orbit.

Fig. 1.

Fig. 1 shows a section of the sarcomatous tumour connected with the exterior layers of the dural sheath of the optic nerve, and apparently originating from it. Its elements are small round cells (diam. $7\mu$); their great bulk is constituted by a nucleus, surrounding which is a very narrow unstained zone. Between

the cells there is a scanty faintly fibrillated intercellular substance. These cells are found lying in rows or clusters between the fibres constituting the outermost part of the dural sheath.

External to the limit of the sheath the cells are closely arranged. Still more distally from the nerve the cells are found infiltrating the fat of the orbit.

Fig. 2.

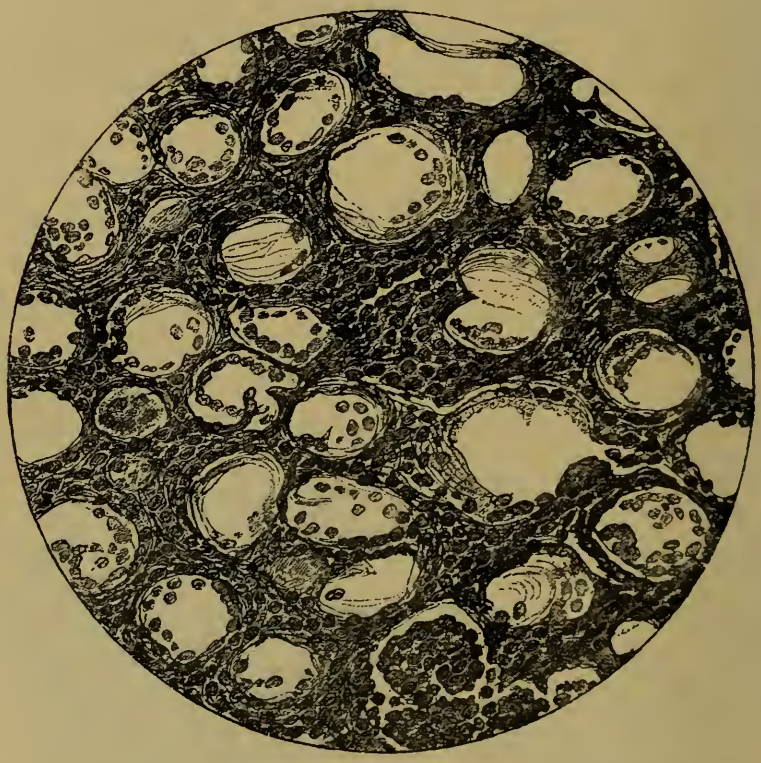


Fig. 2 represents the tumour-elements infiltrating the fat vesicles of the orbit. The white spaces are the fat cells, which have been emptied by ether.

The patient recovered rapidly from the effects of the operation, but the tumour soon recurred, and grew with great rapidity.

On January 12th, 1881, the patient was admitted into the Handel Ward of the Middlesex Hospital.

There was then a solid tumour occupying the orbit and protruding as a large fungoid mass from between the eyelids, which were red and swollen. From the ulcerated surface of the tumour

there was a bloody fœtid discharge. The parotid, submaxillary, and a chain of glands in the left side of the neck were very large, hard, and nodulated.

The man stated that he detected the recurrence about a month after the operation, and that the tumour had grown rapidly.

He was extremely emaciated, and suffered at times much pain in the side of the head. The fungating tumour was dressed with antiseptic lotions, and opium was given in such repeated doses as required, to allay pain and produce sleep.

Gradually the patient became weaker, and on February 16th, 1881, he died.

The following is the report of the *post-mortem* examination :—

*General Appearance.*—Body emaciated; a large fungoid growth protrudes from between the left eyelids. The whole of the left side of the neck and supra-clavicular fossa were filled with nodular masses and diffused secondary growths. The right side of the neck had also some prominent nodules beneath the skin.

*Abdomen.*—The intestines were moderately distended. The mesenteric glands uniformly enlarged and mostly of the size of small beans. On section these glands were white and glistening. A large mass of secondary growths was found around the duodenum and pancreas.

*Thorax.*—The left pleura was adherent everywhere. There were also a few adhesions at the apex of the right pleura. There was no fluid in the pleural cavities.

*Heart*, 10 oz.—The pericardium contained a few drachms of clear serum. The right auricle and ventricle contained a large clot and some fluid blood. The left ventricle contained fluid blood and a small ante-mortem clot. The valves were normal and patent. The aorta was slightly atheromatous.

*The Right Lung*, 27 oz., was large and emphysematous; the bronchi were filled with frothy mucus, and the mucous membrane was injected. The upper lobe was slightly congested; the middle and lower lobes were deeply congested and œdematous. The bronchial glands were enlarged and filled with secondary sarcomatous growth.

*The Left Lung*, 14 oz., was congested throughout, but slightly crepitant and very œdematous.

*Right Kidney*, $6\frac{1}{2}$ oz.; *Left Kidney*, $6\frac{3}{4}$ oz.—Both kidneys were filled with secondary sarcomatous nodules, hard and white on section; these were distributed equally through the cortex and medulla.

*Supra Renals.*—The supra renal bodies were normal.

*Liver.*—Surrounding the portal vein there was a large mass of secondary deposit in the form of rounded tumours. On section they were soft and white, and in the centre there were generally some fine hæmorrhages. In the substance of the liver there were a number of secondary deposits of a similar kind, one the size of a Tangerine orange being situated in the left lobe just beneath the diaphragm.

*Spleen*, 6 oz.—A few small nodules were found in the cortical tissue of the spleen. The organ was congested.

*Brain.*—On removing the skull cap the anterior two-thirds of the surface of the left hemisphere was found to be covered with a thick layer of greenish coagulated lymph. The vessels of the pia mater were minutely injected all over the left hemisphere. On the right side the injection was much less marked. On removing the brain and exposing the orbital plates of the frontal and sphenoid bones, they were found to be very thin, and almost perforated towards the posterior part of the orbit. On lifting up the orbital plates the right or inner wall of the left orbit was found to be deficient; its place was occupied by a soft pulpy mass, which extended into the substance of the ethmoid and sphenoid bones. This was an extension of the growth from the orbit.

*Frontal Lobes.*—The under surface of the apex of the left frontal lobe was soft and of a reddish-brown tint; this softening extended for some little distance into the substance of the frontal lobes. Just outside the softened patch there was a small hæmorrhage into the pia mater, and similar small extravasations were found on the under surface of the cerebellum and pons Varolii.

*Optic Nerve.*—A section of the optic nerves showed that the left was of a perceptibly greater diameter than the right.

*Left Orbit.*—The orbit contained a soft mass of recurrent growth, which nearly filled its cavity.

## ON A CASE OF HYDATID TUMOUR OF ORBIT—PROTRUSION OF EYE—SUPPURATION OF HYDATID CYST—REMOVAL—RECOVERY.

By George Lawson, F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital, and to the Middlesex Hospital.*

In the "Clinical Transactions" of 1876, vol. ix, p. 134, I recorded the case of a patient from whom I removed a large hydatid tumour of the orbit, and gave the report of Dr. Cobbold's examination of the cyst. The case which I have now to relate is another instance of that remarkable affection. The patient was sent to me by Mr. Wilcox, of Aylesbury, who had seen her once or twice previous to her coming to the Hospital.

*History of Case.*—Seven or eight years ago the patient had diplopia, which continued for some time, and only disappeared when the sight of the eye became so defective that it ceased to take cognisance of surrounding objects. Soon after this the eye became slightly prominent. This protrusion of the globe gradually progressed, and with occasional attacks of pain in the orbit. Three weeks before admission the prominence of the eye having greatly increased, Mr. Wilcox punctured the tumour, and some clear colourless fluid escaped. After that there was great pain in and about the tumour, associated with redness and swelling of the lids. The patient was admitted into Moorfields on December 30th, 1881.

*State on Admission.*—There was great proptosis of the right eye, the globe being bulged about three-quarters of an inch from the head. There was swelling and chemosis of the conjunctiva of the lids and globe. The lower eyelid was red, and from the swollen state of the conjunctiva completely everted. The sight of that eye was reduced to a mere perception of large objects. A free incision was made into the tumour through the everted oculo-palpebral fold of conjunctiva of the lower lid, when a

quantity of pus escaped. The finger was then introduced into the wound, and a thick hydatid cyst, about the size of a pigeon egg, was turned out. The walls of the cyst were laminated, of a yellowish-white colour, and of a leathery consistence.

The patient progressed satisfactorily, the globe receded to its normal position, and the patient left the hospital on January 4th, 1882, with the wound almost healed.

# ON A CASE OF LARGE ORBITAL AND INTRACRANIAL IVORY EXOSTOSIS—REMOVAL OF ORBITAL PORTION—DEATH THIRTY-TWO DAYS AFTER OPERATION—NECROPSY.

By John Tweedy, F.R.C.S.,

*Professor of Ophthalmic Medicine and Surgery in University College; Assistant Surgeon to the Royal London Ophthalmic Hospital.*

Two reasons may be assigned for the publication of the following notes: first, cases of ivory exostosis of the orbit are comparatively rare; second, this particular instance was seen by a large number of visitors to the Moorfields Ophthalmic Hospital during the meeting of the International Medical Congress. Those who witnessed the operation will remember its difficulties, and may wish to know its result. The patient speedily rallied from the immediate effects of the operation, and progressed so satisfactorily for a month, that all anxiety seemed to be at an end. Thirty-one days after the operation he suddenly manifested symptoms of cerebral meningitis and died in a few hours. While I regret the fatal termination, I cannot conceal the fact that from the pathological standpoint, and, indirectly, from the clinical, the post-mortem examination furnishes lessons of greater value and importance than those afforded by the living case.

Herbert B., aged 25 years, an excavator, was admitted on July 26, 1881. It was stated that the left eyeball had been prominent for two years, and that the protrusion was steadily increasing. There was, however, no pain either in the eyeball itself or in the parts around.

On examination shortly after admission, the left globe was found to be pushed forwards, downwards, and outwards, by a growth of bony hardness occupying the upper and inner part of the orbit from the mid-orbital ridge to the situation of the lacrimal sac. The movements of the globe were not much

restricted, and the vision was normal $\left(V = \frac{20}{20}\right)$, notwithstanding the presence of well marked optic neuritis, characterised by great œdema of the papilla and enlargement and tortuosity of the veins.

The sight of the right eye was also normal.

After consultation with my colleagues, Messrs. Lawson and Couper, who confirmed the diagnosis of a bony tumour, I resolved to make an exploratory incision, and remove the growth if desirable. This I accordingly did on August 2.

The patient having been put under the influence of ether, I made an incision an inch and a half long through the skin and the orbicular muscle, along the superciliary ridge, and opposite the base of the tumour. The growth was found to be semiglobular in shape and of uniform hardness. It filled the upper and inner part of the orbit in front, and extended backwards as far as the finger could reach. After a brief consultation, in which I had the benefit of the personal experience of Dr. H. Knapp, of New York, I set to work to remove as much of the growth as I could. The periosteum was incised close to the orbital margin, and then stripped off the tumour from before backwards by means of a raspatory. Several ineffectual attempts were made to penetrate the base of the growth, first with a gouge, and afterwards with highly tempered steel chisels and a mallet, but the bone was so hard that no impression could be made on it; the edges of the chisels were soon spoiled. I then had recourse to a drill worked by a powerful Archimedean screw. By means of drills of various sizes I managed to bore a large number of tunnels in various directions through the base of the growth, parallel with the upper and inner walls of the orbit. I then cut through the narrow bony septa, between the borings, with a stout carpenter's chisel. When the chisel had made its way deep enough to act as a wedge, I gave a few energetic blows with the mallet, and succeeded in breaking off the whole of the orbital portion of the growth. When this was accomplished the orbital cavity seemed almost normal in size and shape, though it was obvious that a large mass forming the base of the growth still remained behind. As the operation had lasted nearly two hours, it was deemed unadvisable to prolong it in order to get away the remainder of the growth. The cavity was syringed out with a

warm solution of carbolic acid (1—20), and a caoutchouc drainage tube was inserted. The lips of the wound were then brought together by means of silver wire sutures, and a moist carbolised compress was applied. The patient was carried to bed before he recovered from the anæsthetic.

The piece of bone removed was as large as half a good-sized walnut, and was so dense that fragments of steel from the broken chisels and gouge were firmly imbedded in the surface of section, which was as smooth and bright as polished ivory.

No constitutional reaction followed the severe and prolonged operative procedure. The pulse and temperature did not transcend the normal limits. Sleep was tranquil, and the appetite continued unimpaired.

Next morning, with the exception of some listlessness, the patient's manner seemed unchanged. Then, as on succeeding days, the wound was kept thoroughly drained by means of the rubber-tube, and was syringed out twice a-day with a solution of carbolic acid (1—40). A carbolised compress was, in addition, kept constantly applied over the lips of the wound.

A week after the operation the man got out of bed, and seemed to be in perfect health. The discharge from the wound gradually diminished, and the inflammatory swelling steadily subsided.

Three weeks after the operation there was scarcely any discharge from the wound, and no pain or discomfort. Though the swelling opposite the inner angle of the orbit had grown less, it was noticed that the protrusion and displacement of the globe remained unaltered. The movements were, however, freer than before the operation.

The progress of the case continued to all appearance satisfactory, and altogether devoid of anxiety and trouble, till the morning of September 2, or exactly one calendar month from the day of operation.

On September 1st the man had been exercising as usual in the ward and in the out-buildings of the Hospital, seemingly in perfect health and strength. At 8 P.M. he complained of slight pain at the top of his head. This he himself ascribed to cold. He had a disturbed night, and at 10.30 next morning his temperature suddenly rose to 102° F., and the

pulse went up to 80. The skin was dry, the tongue was clean, and the bowels were free.

I saw the case at mid-day, and found a little increase of swelling about the wound, and thinking that pus might be pent up, I opened up the wound gently with a large probe. I failed to discover any great collection of pus, though a small quantity of dirty-looking shreddy fluid followed the withdrawal of the probe. The wound was gently syringed with the carbolic lotion and the compresses were renewed.

At 9 P.M. there was sudden exacerbation of the headache with occasional rigors. The temperature was 103° F., pulse 60, but very irregular. Ice was applied to the head. Shortly afterwards unconsciousness supervened, and at 11 P.M. profound coma set in, with stertorous breathing, and occasional slight convulsive attacks, equal on both sides. There was no hemiplegia or paralysis of the facial or ocular muscles. The pupils were not dilated, but were equal, and acted to light. At midnight the pulse was 120, full and bounding, and the respiration was laboured. Half-an-hour later the man died. There was no convulsion for about an hour before death.

*Necropsy*, September 3, fifteen hours after death.

Body muscular and well nourished. Above the inner canthus of the left eye was a circular opening which had been occupied by drainage tube, and which led down to bare bone, where tumour had been chiselled off. On removing the calvaria it was found that the greater part of the exostosis was in the left anterior cranial fossa. It had originally sprung from within the left frontal sinus, and had grown chiefly upwards. The roof of the sinus was in part extended over the tumour, and in part absorbed by it. In the frontal sinus were a few semi-transparent polypoid growths. The bony growth reached one inch above the orbital plate of the frontal bone, and extended for that distance into the left anterior lobe of the brain. It measured two inches from before backwards. It also sent a prolongation about half-an-inch long downwards into left side of the nasal cavity, and this portion was broken off the remainder of tumour, apparently at the time of the operation. Backwards, the growth had forced its way into the inner end of the great sphenoidal fissure. The cut surface of the growth had numerous bits of steel embedded in it. The growth was

capable of being slightly moved. There was no marked increase of subarachnoid fluid. There was general basilar meningitis, with a little lymph in the fissure of Sylvius and at the anterior part of the pons Varolii.

The brain in immediate contact with the tumour was in a state of red softening, being semi-fluid, with some puro-lymph on its surface at the summit of the bony mass. The left olfactory bulb was involved in this focus of softening and degeneration. The left optic nerve was stretched across a groove in the growth at the posterior part. The tumour with the adjacent superciliary ridge was removed, and the brain as well as the backs of both eyes and the optic nerves and chiasma were preserved for further examination.

The portion which had been removed by operation was claimed by relatives, who interred it with the body.

The microscopical examination of the optic nerve disclosed the presence of a certain amount of inflammatory cells in the intervaginal space, but without much distension. In the nerve itself were two tracts of inflammation in the midst of otherwise normal tissue (*see* Fig. 3). One of these extended from near the central vessels towards the pial sheath, though without distinct relations to either; the other was smaller, and situated on the opposite side of the central vessels midway between them and the sheath. In these inflammatory areas a few hypertrophied trabeculæ were to be seen, and the nerve-tissue was crowded and obscured by great numbers of nuclei.

*Remarks.*—The practical lesson taught by this case is that it is impossible by the orbital portion alone to judge of the real size and extent of an exostosis springing from the frontal sinus; and, consequently, that other indications must be sought for in considering the feasibility of removing the tumour by operative interference. Though the operation performed in this case may not have shortened life, it would be idle to pretend that it prolonged life.

Should another case of exostosis ever come under my care, I shall endeavour to ascertain the size of the growth and its probable extent within the cranium before deciding to complete the operation.

The difficulty of making any impression on an ivory exostosis with a chisel, saw, or trephine, is so great that several cases have been recorded in which the operation had been abandoned. Keate tried to remove one of these growths with a trephine, but had to desist on account of the hardness, and Sir A. Cooper attempted in vain to saw an ivory exostosis from the edge of orbit.*

Mackenzie† relates that in the year 1843 he witnessed a case similar to those of Keate and Cooper. A gouge, cutting pliers, rasp, and paring-knife were tried on the surface of the exostosis without effect. A chain-saw was then applied, but would not work. Finally, with a Hey's saw a small portion of the growth was subsequently removed, with great difficulty. Ten years later, the exostosis remained exposed in the wound, and bore the marks of the saw "as if the operation had been done but yesterday."

It may interest some to know the sequel of this case. It is given in manuscript in one of the interleaves of the late Dr. Mackenzie's private copy of his work, which is in my possession by the favour of my esteemed friend and teacher, Mr. Wharton Jones, F.R.S.

The rough notes in Dr. Mackenzie's handwriting run thus :—" 26th May, 1858. Five years since last report, patient was admitted into Eye Infirmary; exostosis much more protruded, much more movable, and pus copiously discharged from behind it. Without much trouble it came away. It is much necrosed, and of a very irregular form. The orbit felt entire after its removal, but evidently much displaced downwards, along with the eyeball, which was twisted outwards. The upper lid much shrunk and misshapen. No vision in eye. Cavity filled with lint. Cornea not so opake, I think, as it was. Long diameter of piece $1\frac{7}{8}$ inch. weighed 4 drams 2 scruples and 7 grains. The exostosis seems to have been developed between the inner and outer

* Quoted by Mackenzie, A Practical Treatise of the Diseases of the Eye, 4th ed., Lond., 1854, p. 47.

† *Ibid.*, p. 48.

table of os frontis, leaving a large cavern communicating with the right frontal sinus. The roof of orbit, or rather its lower table, is depressed, along with eyeball. The floor of orbit is half-an-inch below its proper level. Is now free from pain and expresses great relief by removal of exostosis. Piece of carious bone felt near frontal sinus. Parts granulating and healthy. Upper lid much shrunk and displaced."

Other cases have been recorded where the operation had to be given up. Dr. Knapp had to abandon the operation in one case. Seven weeks after the operation, the first five having been passed quietly and favourably, the patient was attacked with symptoms of meningitis of which she died.* Since that case Dr. Knapp has succeeded in removing the tumour on at least two occasions. In the last case an ivory exostosis as large as an English walnut was removed entirely by a process of "subperiosteal enucleation."† The osseous surroundings and connexions of the tumour were cut through with a narrow chisel and the mass shelled out from its periosteal coverings.

As I have already stated I had, during my operation, the benefit of Dr. Knapp's presence and counsel. I was, however, afraid to adopt the plan recommended by Dr. Knapp in so large a growth. The sequel of the case justified my fears. It would have been impossible to have removed the whole of the tumour by the subperiosteal method. Even after death, with the calvaria removed, it was only possible to withdraw the whole of the growth by sawing through the superciliary portion of the frontal bone. Nevertheless, in a demonstrably small exostosis, I readily admit that Dr. Knapp's method offers the best prospect of safe and effectual removal.

* Graefe's Arch. f. Ophth., viii, 1, p. 239, *et seq.*, and quoted by S. Wells, A Treatise on Diseases of the Eye, 3rd. ed, London, 1873, p. 722.

† Archives of Ophthalmology, vol. ix, pt. 4, New York, 1880, pp. 464—470.

## EXPLANATION OF THE FIGURES IN THE PLATE.

Figs. 1 and 2 show the actual size and relative positions and appearance of the growth on its outer and its inner aspects respectively. (The drawings have been inadvertently reversed by the engraver, and seem to represent right side instead of the left).

In Fig. 1 the atrophied shell of the orbital plate is seen forced upwards by the projecting growth. The optic nerve originally lay deeply imbedded in the groove immediately behind the horizontal portion of the orbital plate. The roughened surface below this process, and looking to the right of the figure, represents the surface from which the orbital portion of the exostosis was detached at the operation.

In Fig. 2 a smooth piece of the orbital plate or part of the ethmoidal bone is seen attached to the tumour about the centre. The part above this level was intracranial, and the smooth oval piece to the lower part, and the left side of the figure, lay within the nasal cavity.

Fig. 3 is a representation of the microscopical appearance of a section of the optic nerve. For a description see p. 307.

*Fig. 2.*

*Fig. 1.*

*Fig. 3.*

F Huth, Lith. Edin.

## ON A CASE OF TUMOUR OF THE BRAIN—OPTIC NEURITIS—DEATH—NECROPSY.

By Geo. Lawson, F.R.C.S.,

*Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital.*

The following case was under the care of Surgeon-Major Perry of the Coldstream Guards, and was seen by Sir William Gull, Dr. Hughlings-Jackson, and myself. From the general symptoms and the well marked optic neuritis of one eye, a tumour of the brain was thought probable. The diagnosis was verified by the post-mortem examination which was made by Dr. Greenfield :—

The patient was first seen by Mr. Perry early one morning in August, 1878, and to him I am indebted for the following notes :—He had then had a severe epileptic convulsion, but had recovered from it, and was sitting up in bed and able to answer questions. Within an hour the convulsions recurred. They were very violent ; each convulsion lasted a few minutes, and was followed by an interval of coma, and the convulsions recurred about every twenty minutes. These recurrences continued for about seven hours, when the convulsions ceased, and the patient drifted into deep coma. This state of coma lasted for about three hours. The patient then became restless, and later on delirious and violent. He would frequently try to speak, but could not articulate words. After about twenty-four hours from the first convulsion he fell asleep, and on waking he had recovered consciousness. For the next few days he was kept very quiet, and with the exception that he complained of feeling stiff and bruised, and that his expression was dull and heavy, he seemed pretty well. Previous to this attack the patient had for some time slept badly, and was irritable. He was a man of the most abstemious habits.

In the September following he was sufficiently recovered to be able to be removed to the country, and he was ordered to take bromide of potassium, which he continued for about three

months. He steadily improved in health, and travelled about the country visiting various friends. The patient had no return of the convulsions, but on two occasions between September and the beginning of December, he had two attacks of most violent headache. He described the pain as being almost unbearable.

On January 29th, 1879, he was seen by Sir W. Gull and Dr. Hughlings-Jackson, who diagnosed a tumour of the brain. The patient seemed to get steadily worse; he became weak, and a little unsteady in his gait. He was unable to fix his attention or to read for long.

In April, 1879, I saw the patient. The distant vision of both eyes was normal, but he had considerable loss of accommodative power. His memory he said was defective, and his walk was distinctly unsteady. With the ophthalmoscope there was seen well marked optic neuritis in the right eye; the disc was blurred and swollen, and the veins very large. My diagnosis concurred with that already given.

His unfavourable symptoms continued steadily to increase.

He became more feeble, complained of defect of sight, and towards the end of May his memory was markedly deficient.

On June 3, Dr. Perry states in his notes the patient is very feeble; the sight worse; and he complains of headache.

June 9. Patient very drowsy and difficult to rouse. He remained all day in a condition of semi-coma.

June 14. Is able to speak a few words with difficulty; no headache; has some difficulty in swallowing.

June 19. Condition much the same; talks clearly. Is under the impression that he is by the sea away from home. Pulse 67; temperature normal. The temperature has been taken daily. Occasionally it has been one degree above normal.

June 26. Is not so well; sight very defective; mind confused; his bowels and bladder act involuntarily. Thinks he is in the country.

July 4. General weakness increasing, and is losing flesh rapidly. Appetite good. Sleeps well without the bromide of potassium. Fancies that he is constantly travelling.

July 14. Not so well. At 3.30 P.M. found him almost insensible. Right side of face paralysed. Right pupil dilated and insensible to light. Left pupil contracted. When spoken

to loudly will put out his tongue slowly, and will not withdraw it until again spoken to sharply.

July 15. Spoke a few words in the morning. This afternoon he is in a state of coma, breathing stertorously. Pulse 70. Temperature normal. Pupils the same.

July 16. Has recovered from the state of coma. He thinks that he is abroad. His mind always wandering to places at which he had resided when in health.

July 18. The patient was able to be carried into the garden to-day. Passes his stools involuntarily.

July 30. Has been in a state of semi-coma for fifty-four hours. Pupils continue the same.

July 31. Gradually recovering consciousness.

August 2. Has again become unconscious.

August 3. Is slightly better, and spoke a little, but could not recognise his friends. There is much rigidity of the muscles of the arms. Pulse 62. Temperature 98.

August 6. Has had a quiet night. Spoke at times, and seemed sensible, but did not know where he was.

August 10. Has again relapsed into insensibility, and cannot be roused to speak. Is able to take small quantities of liquid food. The left arm and leg quite rigid, and from time to time there are slight convulsive movements of the left leg.

August 16. Quite insensible. Pulse 40. Extremely weak. Towards evening he rallied. Pulse 58. Temperature 96·6.

August 22. More sensible. Pulse 58. Temperature 97·4. Contraction of flexors of right arm, and slight contraction of flexors of right thigh and leg.

August 24. Passed a very restless night. Face flushed. Temperature 101. Pulse 112. Profuse perspiration. Pupils the same. In the afternoon convulsive movements of the left leg, and with the neck arched back. This condition lasted one hour, during which time his face was much distorted. Pulse rose to 140. From this condition he again passed into coma, in which he continued until he died on August 31.

*Report of the Post-mortem Examination made Twenty-four Hours after death by Dr. Greenfield.*

Only the head was examined. Bones of skull natural.

Dura mater externally appeared somewhat too vascular, the veins also full of blood. Longitudinal sinus full, but quite natural. Pacchionian bodies well marked.

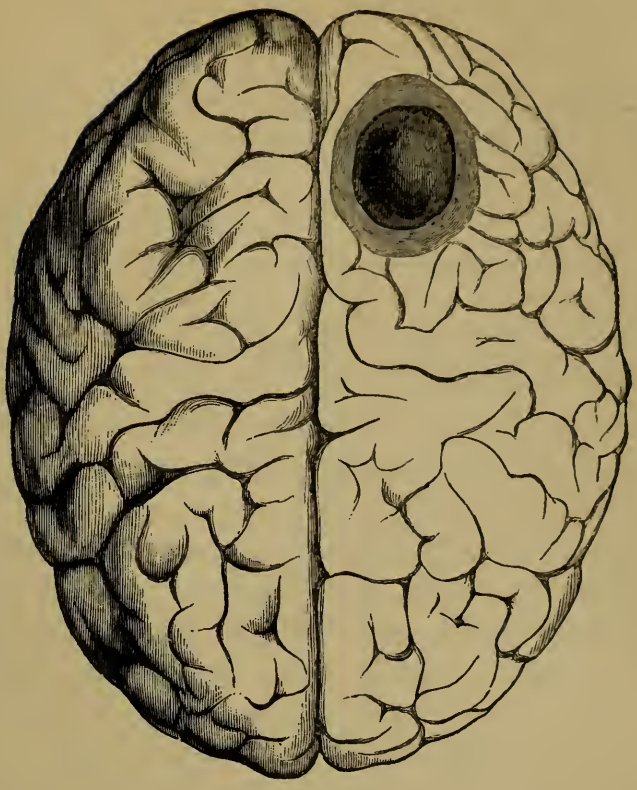
Before removal of the dura mater it was observed that there was greater prominence over the anterior half of the right hemisphere than the left, and an area in the same region where the dura mater was somewhat more opaque than natural, but beyond this nothing abnormal. On attempting to separate the membrane it was found to be firmly adherent to the upper surface of the right hemisphere over a circular area $1\frac{1}{4}$ inches in diameter, near the middle line, and commencing about 1 inch from the tip of the hemisphere. Over the adjacent portion of the hemisphere the dura mater was very slightly adherent to the surface of the brain by a thin layer of sticky exudation; and there were traces of slight recent meningitis (rosy colour of pia mater, dull appearance of arachnoid, turbid fluid in meshes of pia), extending backwards nearly to the occipital region.

Over the left hemisphere dura mater separated readily; no sign of meningitis.

The anterior third of the right hemisphere was to a large extent occupied by a tumour of somewhat ovoid shape, which measured $2\frac{1}{4}$ inches in length, 2 inches in width, and $1\frac{1}{3}$ from above downwards. It weighed nearly 4 ozs. Where it reached the surface it involved the posterior two-thirds of the 1st frontal convolution and a small part of the middle of the 2nd frontal; it was limited posteriorly by the anterior margin of the ascending frontal convolution. Towards the inner (median) aspect of the hemisphere it did not come to the surface, though separated from it by a very thin shell (not more than $\frac{1}{8}$th inch thick) of brain substance.

Below, it encroached upon the anterior part of the right lateral ventricle. No excess of fluid in ventricles. Marked softening of the brain substance surrounding the tumour, especially below and to the outer side.

The tumour itself was remarkably dense and firm, very sharply defined, and readily separated from the surrounding brain tissue, though firmly adherent to the dura mater above. Its surface, smooth, rounded, slightly lobulated, highly vascular, covered by a ramified network of small vessels, from which vessels passed into the surrounding brain tissue. On section it



The diagram represents only the part of the tumour which came to the surface, the darker shaded portion being the area adherent to the dura mater. It is only approximately accurate.

was of somewhat dark fleshy-red colour, rather paler towards the centre; highly vascular; homogeneous and not encapsuled; slightly granular in appearance, but apparently made up of interlacing bands of tissue; very firm to the touch.

The remainder of the substance of the brain appeared in every respect healthy; the centra ovalia pale.

Vessels and nerves at base perfectly healthy, with the exception of left optic nerve, which appeared somewhat flattened, as if wasted.

Cerebellum, pons, medulla oblongata, and upper part of cord appeared perfectly healthy.

W. S. Greenfield, M.D., F.R.C.P.

*September* 2, 1879.

## ON EIGHT CASES OF CONICAL CORNEA, TREATED BY ELLIPTICAL EXCISION OF APEX.

By C. Higgens, F.R.C.S.,

*Assistant Ophthalmic Surgeon to Guy's Hospital.*

The following eight cases of conical cornea have all been treated by the operation which I first saw performed by Mr. Bader, viz., by the removal from the apex of the cone of a small more or less elliptical portion, comprising the whole thickness of the cornea.

The operation is performed by transfixion with a thin Graefe's knife, formation of a small flap, which is picked up with iris forceps and removed with a stroke of the knife in a direction opposite to that in which the flap was made. The anterior chamber is opened pretty freely, but the opening into it is not so large as the wound on the anterior surface of the cornea, the incisions passing obliquely through its structure from within outwards.

The after-treatment consists in keeping both eyes carefully bandaged for a longer or shorter period, according to circumstances. The dressing (wet lint) is changed daily, but neither eye is opened for at least a week.

Iridectomy is performed later if anterior synechia sufficient to close, or nearly close, the pupil occur. The opacity if unsightly is tinted.

Whether the results of my cases compare favourably with those obtained by other methods of treatment I do not know. They are as follows:—

Case 1. Recovered with anterior synechia, but with greatly improved vision. Before the operation the eye could not see well enough to count fingers. Eight months after $V = \frac{20}{50}$, and Snellen $1\frac{1}{2}$ (·5) could be read by the aid of + 12″.

Case 2. Had an anterior synechia, which afterwards separated spontaneously. Vision improved from ability to read Snellen 12 (= 4) by the aid of a small circular opening, to $\frac{20}{200}$, and Snellen $3\frac{1}{2}$ (1·25) at 6 inches without assistance.

Case 3. Went entirely to the bad.

Case 4. Vision was improved from counting fingers to Snellen ·5 at 4 inches, $\frac{6}{36}$ by the aid of −10 D. The pupil was circular and active.

Case 5. Recovered with dense corneal opacity and anterior synechia, requiring iridectomy and tinting. Snellen 1·5 could be read at 6 inches.

Case 6. Vision was greatly improved, but the patient left the hospital before it was accurately tested. The pupil was circular.

Case 7. Recovered with circular movable pupil. Vision was improved as much as could be expected, seeing the condition of the deeper parts of the eye.

Case 8. It was necessary to repeat the operation. On the first occasion a portion of cornea was removed from the side of the cone with but little effect. An anterior synechia resulted, which became detached spontaneously after the second operation. The pupil was circular and movable, vision improved from perception of shadows only to Jaeg. 4 at 2 inches, a result as good as could be expected, seeing the condition of the vitreous.

Case 1. Thursa O., a servant maid, first came under observation at the Central London Ophthalmic Hospital in March, 1872.

The sight of the right eye had been failing five years, but had grown rapidly worse in the last six months. The left eye had been failing two years.

The right eye could distinguish large objects, but could not count fingers.

The left eye read Snellen $2\frac{1}{2}$ at 4 inches.

On April 2, a small portion, including the whole thickness of the cornea, was removed from the apex of the cone in the right eye.

No bad symptom occurred. On the 8th of May there was considerable corneal opacity with anterior synechia.

$$V = \frac{20}{50}.$$

On November 30, nearly eight months after the operation, vision of right eye $= \frac{20}{50}\ c + 12''$, $\frac{20}{50}$; read Snellen $1\frac{1}{2}\ c + 12''$.

The left eye had grown no worse.

Case 2. Sarah G., æt. 30, a lady's maid, came under treatment at Guy's Hospital December 7, 1876.

Believed that she had been getting near sighted for some considerable time. Ten months before date had accidentally covered the left eye, and found that she could not see with the right. No lenses improved sight, but $V = \frac{20}{50}$ with small circular opening, without it large objects could be seen indistinctly. Left eye $V = \frac{20}{200}\ c - 6''\ \frac{20}{50}$.

Nine months later right eye could make out Sn. 12 at 4 inches when using stenopeic plate.

The left eye by the same means read Sn. $3\frac{1}{2}$ at 6 inches.

November 27, 1876. A small elliptical piece of cornea, including its whole thickness, removed from the apex of the cone in right eye.

The wound healed firmly. On January 1, 1877, there was an opacity of the cornea, rather above and to the outer side of the pupil, the margin of which was adherent to it.

A month later, the anterior synechia had given way from action of the iris unaided by atropine; the pupil was circular and freely movable; the corneal opacity remained about the same.

Right eye, $V = \frac{20}{200}$. No improvement with lenses. Read Sn. $3\frac{1}{2}$ at 6 inches easily.

Left eye, $V = \frac{20}{70}$ not improved by lenses, but rather better with stenopeic plate Sn. $2\frac{1}{2}$ at 10 inches.

Case 3. Lizzie M., æt. 22, no occupation, has done a great deal of fine sewing and much reading; first came under treatment in January, 1879. She had been a little near sighted as long as she could remember, but during the last five years she had noticed that she was getting rapidly worse. The right eye failed faster than the left, and at the end of three years was nearly blind; the left could still distinguish a person across a fair sized room, and could see to read a newspaper with difficulty at about 6 inches. During the last two years the sight of both eyes had remained about the same.

The right eye could make out Sn. 4 $c -\frac{1}{12}$ at 8 inches. With horizontal stenopeic slit Sn. 3 at 16 inches, through small circular opening Sn. 2·25 could be read at 8 inches, and V $= \frac{6}{36}$.

The left eye could read S. 1·25 at 5 inches without aid, with small circular opening V $= \frac{6}{24}$.

On January 13 a small elliptical piece, including the whole thickness of the cornea, was removed from the apex of the cone.

Ten days later the eyelids were red and swollen, and the cornea was found to be infiltrated with pus.

The central portion of the cornea disappeared, and was replaced by iris and cicatricial tissue, a ring of clear cornea remaining.

Subsequently iridectomy was performed downwards and outwards. The pupil became blocked by inflammatory material. The eye retained perception of light.

Case 4. Lucy R., æt. 18, came under treatment in December, 1879. Right eye could not count fingers; with stenopeic plate could read a letter here and there of Sn. 4. Left eye could read Sn. 4 at 4 inches without aid, Sn. 6 at 4 inches with stenopeic, and V $= \frac{6}{36}\ c\ -$ 5·50 D.

December 15, 1879. Portion of cornea, including its whole thickness, removed from apex of cone.

A fortnight later the wound was firmly healed, and the pupil circular and active.

On February 1, 1880, six weeks after the operation, a small

linear opacity of the cornea rather above the centre of the pupil, remained. The eye could read Sn. 1·5 at 4 inches without aid, and $V = \frac{6}{36} c - 10$ D.

Case 5. Joseph J., æt. 21, draper's assistant, came to Guy's Hospital February 19, 1880. No history was taken. There was divergent strabismus of the right eye with well marked conical cornea; the eye could barely count fingers at a few inches. Readjustment was performed.

Four days later the conical cornea was treated by removal of a portion of the apex of the cone, including the whole thickness of the cornea. Recovery was slow, but eventually took place, a dense corneal opacity with shallow anterior chamber and anterior synechia remaining.

Five months later the opacity was tinted and iridectomy performed.

A month after, the eye could read Snellen 1·5 at 6 inches.

Case 6. Hildric F., æt. 28, a clergyman, first came August 2, 1880. Left eye well marked conical cornea, some opacity at apex of cone, sight gradually failing; two years could count fingers at a foot.

Right eye failing eighteen months. Could read Sn. 5 at 6 inches. $V = \frac{6}{12}$.

August 9, 1880. A small portion, including whole thickness of cornea, removed from apex of cone.

August 23. Anterior chamber re-established, but still some leakage; pupil circular. No anterior synechia; vision not tested. The patient was anxious to go away, and was not seen afterwards, but wrote some weeks later to say that the eye had quite recovered, and he could see fairly well with it.

Case 7. Annie S., æt. 37, admitted January 17, 1881. Twelve years ago first noticed gradually increasing dimness of sight in the left eye. Lately the right eye has begun to fail, which she attributes to over use.

The left eye sees shadows. There is well marked conical cornea. The right eye $V = \frac{6}{9}$ barely; no improvement with + or -- sph. or cyl. The apex of the cone was removed, as in the other cases.

March 21. Wound soundly healed; no synechia; cone rather large; central opacity. Counts fingers at 3 feet.

September 27. Left cornea slightly conical, central opacity; makes out a letter here and there of Jaeg. 20 at 10″. Pupil circular, movable. There is a large posterior staphyloma. Patient now states she never could see with the left eye.

Case 8. Phillis H., æt. 42, admitted February 21, 1881. Twenty years ago discovered that the sight of the right eye was very defective; she has been unable to read with it since first discovering the defect; does not think there has been much change for many years.

The centre of the cornea is occupied by a very pointed cone, the apex of which is opaque; there is some floating opacity in the vitreous. The eye has perception of shadows only. The left eye has begun to fail in the last few years; the cornea is slightly conical; there is some floating opacity in the vitreous; the eye reads Jaeg. 6 at 6 inches.

A portion of elliptical shape, including the whole thickness of the cornea, was removed from the side of the cone in the right eye.

March 14. Wound soundly healed; small anterior synechia; counts fingers, and can tell one from the other.

April 11. Vision not improved since last report; cone still very prominent; large elliptical piece removed from its apex.

July 4. Reads Jaeg. 4 at 2 inches; the synechia which resulted from the first operation has separated, and the pupil is circular; vision is not improved by lenses.

## ON A CASE OF SYMPATHETIC OPHTHALMIA.

By Anderson Critchett, M.A. (Cantab.),

*Ophthalmic Surgeon to St. Mary's Hospital.*

As the subject of sympathetic ophthalmia is now exciting considerable interest, especially with reference to the incubation period of the disease, and to the relative immunity which can be obtained by removal of the injured eye, the following case may serve to throw some light upon these debatable points:—

J. P., a gamekeeper, æt. 30, was struck in the right eye by a stray shot on November 3, 1881, which penetrated the coats of the eye in the ciliary region. He was taken on the same day to a provincial hospital, where he remained for a fortnight under palliative treatment. On November 17th he was admitted under my care at St. Mary's Hospital. The injured eye was found to have received a small circular wound as described above, and it was evident that an escape of vitreous had occurred, causing the tension of the globe to be minus. The pupil was irregular, a portion of the iris being drawn up and involved in the wound. On ophthalmoscopic examination, only a faint reflex could be obtained. The patient did not complain of pain, and there was but slight evidence of inflammatory action. The appearance of the left eye was in all respects healthy, and the vision, which was accurately tested, was found to be normal; whilst the ophthalmoscope revealed no trace of optic neuritis. On the following day the right eye was enucleated. On examination, the track of the shot could be accurately traced, from its entry at the ciliary region, by the presence of a thin line of blood-clot extending through the vitreous to within a short distance of the corresponding point on the opposite side of the globe, where the shot itself was found, having impinged upon, but not penetrated the retina.

The patient was discharged on November 23, his left eye being re-tested, and found to be in all respects as healthy as

it was on his admission. He was cautioned to use the eye sparingly, and to avoid strong light for some weeks to come. On December 3, *i.e.*, fifteen days after the operation, and exactly a month from the date of the accident, he first noticed that distant objects appeared to be slightly misty and indistinct. His limit of vision lessened daily for about a week, when he could only distinguish large objects at a distance of a few yards. The eye then became red and painful, the condition both as regards vision and inflammation varying somewhat from day to day till the end of March, when the more active symptoms subsided, but the sight remained so far impaired that shadows only could be distinguished. The gravity of these symptoms was not sufficiently appreciated by the patient, who attributed them to an accidental cold; and, as he was at a considerable distance from London, he did not again seek my aid till the 15th of May, when he was re-admitted to St. Mary's Hospital.

In the left eye well-marked signs of sympathetic ophthalmia had set in, though modified in some degree by the rapidity with which it had occurred. The pupil was completely occluded, and the iris was slightly thickened and bulging, though it had evidently not undergone the complete structural metamorphosis which often occurs in strongly-marked cases of this type. The anterior chamber was extremely shallow, and the tension of the globe was + 2. For the relief of this latter symptom, and in the hope of restoring free communication between the anterior and posterior chamber, an iridectomy was performed on May 16th. By making slow and gentle traction on the iris, I was fortunately able to detach it from its adhesion to the anterior capsule and to remove a portion corresponding to the size of my incision in the corneo-sclerotic junction, leaving a clear black aperture. On May 20th the patient was able to count fingers at the distance of 12 inches. At the present date (June 2nd), he can distinguish faces, the tension is normal, the artificial pupil remains clear, and the anterior chamber has almost resumed its natural proportions.

It is worthy of note, that the presence of a foreign body

within the eye considerably increases, as in this instance, the gravity of the case. The isolated instances of gun-shot injury which have come before my notice, where the injured eye has not been removed, probably owed their immunity from subsequent trouble in great measure to the facts that the shot did not wound the ciliary region, and that it went with sufficient force to traverse the globe and lodge in the surrounding tissues.

Until a comparatively recent period, a strong and apparently well-grounded impression prevailed amongst ophthalmologists that when the injured globe was removed, before any evidence of irritation could be noticed in the other eye, complete freedom from sympathetic ophthalmia might be confidently predicted. This comfortable doctrine recently received a rude shock at the hands of Mr. Nettleship, who has conclusively shown that painful exceptions to this rule occasionally occur; and the case which I have quoted above strongly tends to confirm that view. The practical inference which may be drawn from this and similar cases is, that grave danger can result from even a short delay, and that an emphatic decision should, if possible, be at once arrived at as to the necessity which exists for removal of the injured organ, since it is evident from the present case that this peculiar form of sympathetic inflammation must have made an insidious commencement within fifteen days from the date of the accident.

# ON SYMPATHETIC OPHTHALMITIS FOLLOWING EXTRACTION OF CATARACT.

By W. J. Milles, F.R.C.S.,

*Senior House Surgeon, Royal London Ophthalmic Hospital.*

Sympathetic Inflammation, resulting from the extraction of a senile cataract, is happily such a rare disaster that it scarcely suggests itself, when considering the risks of the operation. It has fallen to my lot, as House Surgeon, to have had under observation the unusually large number of eleven cases of undoubted sympathetic inflammation due to the above cause. One of my reasons for giving an account of these cases is that often more may be learned from the disasters than from the successes of surgery.

I have reduced my notes to as small a compass as is consistent with a description of the important points; a minute account of each individual case at the various stages of the disease would have involved an unnecessary repetition.

All the patients were fairly good subjects for operation; the cataracts were not complicated by any other disease of the eyes; the pupils were active, projection good, tension normal.

Case I.—*Extraction of Cataract. Puncture and Counter-puncture in Sclerotic. Prolapse of Iris. Sympathetic Ophthalmitis in three months.*

Mary W., æt. 52, gave a history of failure of the right eye for seven years. She had a mature cataract in this eye, with active pupil, normal projection, and normal tension. Left, immature cataract.

September 25, 1881. *Right.* Modified Graefe extraction, puncture, and counter-puncture in sclerotic, the latter rather far back. The iris was torn at the outer part during the operation. Patient was discharged in three weeks' time without having had any pain.

Readmitted March 3, 1881, with a history of pain in right eye since her discharge from the hospital, and slight uneasiness in the left for the last week.

In the right (exciting eye), there was keratitis punctata, a flattened prolapse of iris, and a firm opaque capsule, and some posterior synechiæ. V = J. 19 c + 16 d. T.—.

In the left (sympathising eye), keratitis punctata was more marked, iritis with a large quantity of lymph in the pupil.

V = counts fingers at 8". T.n.

During the two following months she was under observation, the right eye quieted down, though the keratitis punctata never completely disappeared; the iris became discoloured and drawn backwards in its lower half. The globe was tender at times. T. — always. Vision remained the same.

In the left eye the quantity of lymph gradually increased, till a thick layer covered the anterior capsule of the lens; large masses projected forwards from the iris, cornea was covered with dots, anterior chamber deep. T.n.; V = hand reflex. Pain was at times very severe, especially in the left eye.

Case II.—*Extraction of immature Cataract. Sympathetic Ophthalmitis in eight months. Iridotomy in Exciting Eye, resulting in Normal Vision. Sclerotomy in Sympathising Eye.*

Sarah H., æt. 63.

*Left* counts fingers at 8''; cataract nearly mature.

*Right.* J. 10 c + 4 d.; cataract immature.

May 10, 1880. *Left.* Modified Graefe extraction; a small quantity of soft matter left behind.

Patient discharged at the end of three weeks, with an opaque capsule, and still some soft matter in pupil.

Readmitted January 31, 1881. Right had been getting dim, with some pain during the last fortnight.

*Left.*—Anterior synechiæ of iris and capsule to wound, deep anterior chamber; triangular mass, irregular and punctiform at its edges, on posterior surface of cornea. T. +.

*Right.*—Serous iritis with keratitis punctata. T. +.

In the right (sympathising eye) the pupil dilated slowly, but contracted again through the increase of the lymph deposit. The eye became glaucomatous and very painful, and in August,

1881, sclerotomy was performed; but the wound bulged, and though the pain ceased and all active inflammatory symptoms disappeared, perception of light was completely lost.

In the left (exciting eye) the iritis and mass of lymph had completely gone at the end of two months. An iridotomy was performed on March 17, 1881, with some loss of vitreous, and in the following August the vision was as follows:—

c + 12 D $\frac{20}{20}$ c + 16 D., J. 1 at 8″.

I saw this patient only a short time back, and found that the sight in this eye was still the same.

Case III.—*Extraction of Cataract. Suppurative Irido-Cyclitis. Sympathetic Ophthalmitis in three months.*

Thomas C., æt. 56.

January 27, 1881. *Right.* Modified Graefe's extraction, section wholly corneal, operation uncomplicated. On the third day the wound was infiltrated, anterior chamber soon became filled with puro-lymph, and suppurative irido-cyclitis followed. At the end of two months the eye was quiet, but partially shrunken, T.−1; perception of light. Three months after the operation patient was readmitted with sympathetic iritis in the left eye.

The right (exciting eye) was much in the same condition as at the time of his discharge from the hospital a month ago. The corneal section was depressed, cornea was small, globe slightly shrunken, and not tender on pressure; the iris was drawn up and atrophied. Bare perception of light. T.−1.

In the left (sympathising eye) the posterior synechia yielded to atropine, but soon the iritis became more severe. Keratitis punctata appeared at the end of four months, and masses of lymph filled up the pupillary space. At the end of six months there was a deposit of lymph, covering the lower half of the cornea, and the iris was barely visible, owing to the muddy condition of the aqueous. T. + 1. Sees shadows.

Case IV.—*Preliminary Iridectomy. Extraction of Cataract. Sympathetic Ophthalmitis in six weeks. Vision in Exciting Eye good for some months, gradually failed.*

Jane B., æt. 63.

May 31, 1881. *Right.* Modified Graefe extraction, a preliminary iridectomy having been performed six weeks previously. (The notes of the operation have been mislaid.)

Readmitted July 8, 1881. Patient had experienced some pain in the left eye for the last month.

*Right* (exciting eye). Fine membrane in pupillary space, section corneal, some posterior synechiæ. $V = \frac{20}{50}$ c + 10·5 d., J. 6, c + 15·5 d.

The vision remained as above for two months, when the capsule contracted, vision becoming only $\frac{20}{200}$. The capsule was needled, but the opening closed again.

It was thought advisable not to perform any further operation on this eye till the sympathising eye had quieted down. In six months' time, owing to drawing up of the pupil, V = counting fingers at twelve inches.

In the sympathising eye (left), almost exactly the same changes occurred as in the last case, serous iritis, keratitis punctata; finally a large deposit of lymph. At one stage a number of vesicles appeared on the anterior surface of the cornea. Tension variable from T.n. to T. + 1.

CASE V.—*Extraction of Cataract. Suppurative Irido-Cyclitis. Shrinking of Globe. Sympathetic Ophthalmitis in seven weeks.*

Thomas R., æt. 57.

June 1, 1881. *Right.* Modified Graefe extraction; the operation was uncomplicated. The sequence of events is almost identical with Case III. Suppuration commenced at the corneal wound, suppurative cyclitis followed, and the globe shrank. On being readmitted four months later, the patient stated that the right eye had remained painful since he left the hospital, and that the left had become irritable and painful seven weeks after the operation.

The right (exciting eye) was found to be shrunken, with a puckered corneal scar, painful and tender. No perception of light.

The left (sympathising eye) had shallow ant. chamber; numerous posterior synechiæ, which would not yield to atropine;

large vessels in iris, keratitis punctata, &c. Two months later the whole cornea was covered with dots, the pupil was occluded by lymph, T + 1, and V = shadows.

CASE VI.—*Extraction of Cataract. Entanglement of Iris in corner of Wound. Sympathetic Ophthalmitis in nine weeks. Good Vision in Exciting Eye.*

William T., æt. 72.

June 28, 1881. *Left.* Modified Graefe extraction. The cataract was mature, but the pupil was sluggish previous to the operation. When the patient was discharged at the end of three weeks, an entanglement of iris was noticed at each corner of the wound.

On readmission four months after, he was suffering from sympathetic ophthalmitis, the first symptoms of which had appeared nine weeks after the extraction. The exciting eye had been perfectly quiet.

*Left* (exciting eye) section corneal, small entanglement of iris in each corner of wound; pupil active; no ciliary injection. V c + 11 d = $\frac{20}{70}$. The eye remained in the same condition, and two months later V c + 11 d = $\frac{20}{50}$, c + 15 d. J. 10.

*Right* (sympathising eye) underwent the usual changes of serous iritis; dots on cornea; variable tension. Vision finally shadows.

It is remarkable that no reactive changes whatever occurred in the exciting eye, the only apparent cause of the sympathetic ophthalmitis being a small entanglement of iris.

CASE VII.—*Extraction of Cataract. Suppurative Irido-Cyclitis. Sympathetic Ophthalmitis in eight weeks. Excision of Exciting Eye.*

There is great similarity here to Cases III and V.

Elizabeth T., æt. 68. The sight in the left eye had been failing for twenty-five years. There was a history of pain in the eye for the last four months, but the notes state that she had active pupil; field of V. good, and saw shadows. The right had an immature cataract.

September 30, 1881. *Left:* modified Graefe extraction, some soft matter being left behind. A very slow painless form of suppurative irido-cyclitis ensued, which ultimately cleared up, leaving a partially shrunken globe, and bare perception of light.

Patient readmitted two months later, with history of pain in right eye eight weeks after the operation.

*Left* (exciting eye) was considerably shrunken and tender; the corneal scar was puckered; bare perception of light. T.—1. It was enucleated.

*Right* (sympathising eye) had numerous posterior synechiæ, which yielded to atropine, but soon reformed; dots were noticed six weeks later. At first there was great pain, but it soon ceased. Patient was under observation for five months. Tension had been variable, often T.+1, cornea finally covered with dots; shallow anterior chamber, pupil contracted and occluded by lymph. V = shadows. Pain considerable at times.

Case VIII.—*Extraction of Cataract. Suppurative Iritis and Entanglement of Iris. Sympathetic Ophthalmitis in six weeks.*

Hannah S., æt. 73.

27th May, 1881. *Left.* Modified Graefe extraction. Operation uncomplicated, followed by iritis, and at the end of three weeks by hypopyon, which very slowly disappeared.

On readmission in two months' time, she stated that she had had some pain in right eye six weeks after the operation.

*Left* (exciting eye). There was a small entanglement of iris at inner corner of the section, with adhesion of capsule to middle of it. The pupil was drawn up. T.—. V = shadows.

*Right* (sympathising eye). Serous iritis. Keratitis punctata. T.+1. V = shadows.

This patient was under observation for several months, during which period she was subject to attacks of great pain in the sympathising eye, the tension of which was often high. The exciting eye finally had no perception. T — 1; globe slightly tender. In sympathising eye pupil gradually closed; ant. ch. became shallow; iris swollen, with vessels visible on its surface. V = shadows. T. + 1.

CASE IX.—*Extraction of Cataract. Counter-puncture in Sclerotic. Sympathetic Ophthalmitis in three months. Iridotomy in Exciting Eye.*

H. B., æt. 73.

March 9, 1880. *Left.* Modified Graefe extraction elsewhere. Patient says she could see well after the operation. Three months later the sight in both eyes failed rapidly, the right becoming very painful, and remaining so up to the time of her admission into Moorfields Hospital, on September 19, 1881. Her condition was as follows :—

*Left* (exciting eye). The operation scar reached far into the sclerotic on the inner side; pupil was drawn up and closed by a dense membrane; good perception of light. T.n.

*Right* (sympathising eye). Cornea dull, especially in its lower half and posterior surface; anterior chamber very shallow; pupil occluded; iris sodden and bombé. No perception. T. + 3.

An iridotomy was performed on the left eye; iris was found to be tough, and did not gape satisfactorily. Six weeks later the operation was repeated, and a good permanent opening was obtained. At the end of three months the patient was able to read J. 20 c + 15 d, with exciting eye.

The sympathising eye was painless, but still glaucomatous. No perception of light.

CASE X.—*Extraction of Cataract by Scoop. Closed Pupil. Iridotomy. Sympathetic Ophthalmitis in fifteen months.*

N. C., æt. 61.

April 13, 1880. *Left.* Modified Graefe extraction; lens did not present, and was removed by the scoop, with loss of a small quantity of vitreous. Iritis followed; the pupil became closed and drawn up.

Four months later an iridotomy was performed with a broad needle; a clear central pupil resulted, which, however, closed again in a short time.

After an interval of nearly a year the patient came to the hospital on account of pain in the right eye during the last three weeks.

The right eye was suffering from well marked sympathetic

ophthalmitis. The cornea was dull, and covered with dots aqueous muddy; iris greatly swollen. T.n.

The patient had attacks of pain in this eye during the next few months. T. + 2 at times; keratitis punctata remained constantly present.

*Left* (exciting eye) was not painful or tender. Pupil was drawn up and occluded. Another iridotomy was attempted, but was followed by great reaction, and at the end of a fortnight the opening had completely closed.

The tension of exciting eye became minus, with bare perception of light.

Case XI.—*Extraction of Cataract. Iritis and Closed Pupil. Sympathetic Ophthalmitis in eight weeks, without Pain.*

George P., æt. 76.

December 24, 1881. *Right.* Modified Graefe extraction, followed by a slow painless form of iritis, with lymph filling up the pupil.

Patient was readmitted in three months. He had only noticed slight uneasiness in the left eye, commencing eight weeks after the operation.

*Right* (exciting eye). The pupil was drawn up and occluded by a very dense membrane; no incarceration of iris. The capsule was adherent to the wound; dots over the whole cornea, and aqueous muddy. T.n.

*Left* (sympathising eye). Cornea steamy; iritis; adhesions slowly yielding to atropine. T.n. Dots noticed in a fortnight.

During two months he had no pain in the eyes. In the exciting eye the keratitis punctata resulted in a large plaque; there was very little injection, and no tenderness. T.n. V = shadows. In the sympathising eye the pupil was kept fairly dilated with atropine, aqueous became very muddy; dots increased; anterior capsule of lens was covered by exudation. T.n. V = shadows.

In all cases the treatment (of the sympathetic ophthalmitis) consisted in confining the patient to a dark room, usually with a black bandage over the eyes; in dilating the pupil as long as possible with atropine; and in alleviating

pain by leeches, blisters, and morphia. A course of mercury was tried in a few instances, but with no apparent influence on the progress of the disease.

Perhaps the best method of making a useful comparison of the salient points of these cases, will be to place their leading characteristics under a few important headings.

*Sex and Age.*—The patients were five men and six women, their ages ranging between 52 and 76, the average age being 65.

*Operation.*—Graefe's modified linear extraction was performed in each case. The extremities of the section were within the cornea, except in Case 1, where puncture and counter-puncture were in the sclerotic, and in Case 9, where counter-puncture was far back in the sclerotic.

*Accidents during the Extraction.*—In six cases the operation of extraction was uncomplicated. In two some soft lens matter was left behind. In one the iris was partially torn from its attachment. In one the cataract had to be removed by the scoop, with slight loss of vitreous. In one the notes of the operation have been lost.

*Results from Extraction.*—As regards the condition of the exciting eye after the operation, in two instances there was simply a small incarceration of iris in the corners of the corneal section though with good vision. In one a dense membrane and posterior synechiæ formed as a result of iritis; in two iris and capsule were adherent to the wound; in two more the pupil was drawn up and closed by membrane; in one suppurative iritis of a painless character resulted; and in three instances suppurative irido-cyclitis occurred, and was followed by shrinking of the globe.

*The interval between the extraction and the onset of sympathetic ophthalmitis* was, as a rule, two to three months. The shortest period was six weeks, the longest 15 months.

*Frequency of Keratitis Punctata.*—I was especially careful in watching for this symptom, as I have been struck with the fact that it almost invariably occurs in sympathising eyes. Sometimes it may require a very minute examination to detect the dots; in one case I was able to see them with a

Coddington lens some days before they were visible to the naked eye.

Dr. Brailey from his researches on the pathology of sympathetic ophthalmitis is of opinion that there is always some histological evidence that the disease begins as uveitis serosa, with keratitis punctata, however well marked the uveitis maligna may be at the later stages. From a clinical point of view these cases tend to confirm his statement.

I find from my notes that keratitis punctata was invariably observed in the sympathising eye, usually at an early period. Case 9 is an apparent exception; but it is to be noticed that this patient was not seen till 15 months after the commencement of the disease, and that distinct traces of the keratitis punctata were left in the form of an opacity on the posterior surface of the cornea.

The dots certainly occur at an early stage of the disease; they remain not for weeks, but for months (*see* Cases 2, 4 and 8), and have a tendency constantly to increase in numbers, till they often coalesce into a large plaque; this plaque may ultimately be absorbed, leaving only a few traces behind.

It is interesting to note that in three instances dots were present in both eyes (Cases 1, 2, and 11); they always occurred in the exciting eye first. It shows that occasionally some of the pathological changes that occur in the sympathising eye may be present in the exciting eye.

*Variable Tension.*—In the sympathising eyes the tension varied from time to time; one day it would be normal, another day increased. This variation in the degree of tension is, I believe, one of the characteristics of sympathetic inflammation. I find that in Case 2 only was the tension normal throughout, and it is noticeable that this patient never had any pain or tenderness in the eyes.

*Results as regards Vision.*—It is an interesting and remarkable circumstance that several of these patients retained excellent vision in the exciting eye.

In Case 2, thirteen months after the sympathetic ophthal-

mitis had commenced, the patient was able to read with the exciting eye $\frac{20}{20}$ and J. 1, with correcting glasses.

A parallel case to this is described by Dr. D. Webster.*

Case 6 is nearly as good. He was able to read $\frac{20}{50}$ and J. 10 with the exciting eye five months after the commencement of the sympathetic disease.

In Case 4 the patient's vision in the exciting eye (after three months) was $\frac{20}{50}$ and J. 6 with glasses. The opening in the capsule, however, slowly closed, and vision diminished. Two other patients after eight months and twenty months respectively, were able to read J. 19 and J. 20 with the exciting eye.

The severity of the sympathetic ophthalmitis bore no proportion to the damage done to the exciting eye.

What the final result may be to the sympathising eye it is difficult at present to say. Though some of the patients have returned to the hospital after an interval of several months, active changes were still going on. Under these conditions, of course, any operative interference would have been worse than useless. In one case a sclerotomy was performed on a very painful glaucomatous eye; it was successful in so far that the pain was relieved, though the result to the eye was not satisfactory.

I wish, in conclusion, to acknowledge the courtesy of the various members of the surgical staff in placing the above cases at my disposal.

* Trans. Amer. Ophth. Society, vol. iii, part i.

## ON A CASE OF EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA, MODIFIED BY THE PRESENCE OF A CILIO-RETINAL ARTERY.

By Arthur H. Benson, M.B., F.R.C.S.I.,

*Assistant Surgeon, St. Mark's Ophthalmic Hospital, Dublin; Lecturer on Ophthalmic and Aural Surgery in Ledwich School of Medicine, Dublin.*

Since 1859, when von Graefe made his memorable observation with the ophthalmoscope, which for the first time explained the true cause of a series of cases of sudden blindness, which had before that been amongst the hidden mysteries of disease, embolism of the central artery of the retina has attracted the attention of all ophthalmologists. A vast number of cases has since been placed on record showing much variety in the degree and position of the obstruction, but with symptoms and ophthalmoscopic appearances so uniform and unique that the diagnosis can now in most cases be made without difficulty.

It would, therefore, seem unnecessary for me to add to this record any additional description, nor would I venture to do so, but that one of my cases presents some peculiarly interesting points which make it differ from any others which I have myself seen, or of which I can find any record.

Since reading Mr. Nettleship's remarks on the occurrence of cilio-retinal blood-vessels (vol. ix, part II, p. 161, of these Reports, 1877), I have been on the watch for cases to test the accuracy of his "general statements," Nos. 9 and 10, where he says, "The presence and size of this communicating vessel does not appear to bear any relation to disease;" and again, "no practical significance can at present be attached to this peculiarity in the blood supply of the yellow-spot region of the retina."

It seemed an obvious supposition that if these vessels ran

the course which he described, in the event of an embolus lodging in the trunk of the central artery of the retina, the vessel deriving its blood from the ciliary arteries might still continue to supply that portion of the retina to which it was distributed, and save it from sharing in the general destruction attendant upon a total occlusion of the efferent artery.

Since then I have seen eight cases of embolism of the central artery of the retina, in all of which the peculiar ophthalmoscopic appearances were present, but in none of them was there any indication of the presence of a cilio-retinal vessel, except in the last, the notes of which I here give, and from which I took the accompanying ophthalmoscopic drawings.

M. F., a young lady, aged 16 years, was brought to me on 26th April, 1882. She stated that three days previously whilst quietly at her devotions she observed her left eye become suddenly stone blind. After a few hours the sight somewhat improved, so that she could make out the position of large objects. When I saw her she could see my hand moving, but could not count fingers at any distance. The pupil was slightly dilated and sluggish. The tension was normal. She had experienced no premonitory auras.

The ophthalmoscope showed a considerable area of the retina to be in a condition of ischæmia. The region of the macula was marked by the characteristic cherry-red spot, situated in an area of pale greyish-white œdematous retina. The disc was hazy as to its margins, and was surrounded by an area of œdematous retina on all sides except that towards the macula, in which direction it was normal in appearance.

The œdema, which was very intense immediately round the macula, terminated abruptly by a wavy line slightly to the nasal side of the yellow spot, the line of demarcation diminishing in precision as it curved round to touch the disc above and below, leaving an oval area of perfectly healthy-looking retina existing between the yellow spot and the disc.

The œdema elsewhere faded off into the non-œdematous peripheral retina. The vessels of the retina were not much altered from the normal; the arteries were rather smaller, and

the veins rather larger than the normal, but no empty or thread-like vessels could be observed.

The flow of blood through the veins seemed rather stagnant, and in the large superior temporal branch where it bent over the artery, a distinct difference in the flow could be observed, the blood moving in a visibly incomplete stream suggesting the idea of a waterfall. Pulsation in most of the vessels could easily be produced by pressure. Some of the vessels were seen with accentuated distinctness over the grey œdema, whilst others again were hidden by the opaque nerve fibres.

A *cilio-retinal* vessel was observed springing from the outer side of the disc, and running towards the yellow spot through the region of healthy-looking retina.

To that vessel, no doubt, was due the fact of that portion of the retina being free from œdema.

Fatty degeneration of the retina seemed to have already commenced, and some brightly refracting specks were visible especially at the nasal side of the disc, where the œdema was not intense. One small hæmorrhage was to be seen in the œdematous retina above the macula.

The field of vision was very much restricted.

A careful examination of the heart failed to detect any evidence of disease, nor was there any history of rheumatic fever or other constitutional taint. She had suffered from infantile paralysis, and was still lame on the right leg, but in all other respects she was in excellent health.

*Treatment.*—It seemed pretty certain that whatever had been the seat of obstruction, the circulation was, when I first saw the case, for the most part re-established, though not in its entirety, for the arteries were still small, and the tension of the blood in the veins decidedly low.

Judging by former experience, and by the recommendations of all writers with whom I was acquainted, I anticipated no return of useful vision; for though Dr. Wood-White and Dr. Eales had lately recorded cases in which vision had been recovered, yet in this case so long a time had elapsed since the accident with so little improvement, that I was prepared for the worst result. However, the circulation having been pretty well re-established, it occurred to me that the indication was to keep up the nutrition of the retina until such time as the œdema

would be absorbed. The continuous electrical current had been attended with such benefit in the hands of Mr. Gunn ("Roy. Lond. Ophth. Hosp. Reports," vol. x, part 2, p. 161) in cases of atrophy of the optic nerve, that it seemed reasonable to expect its influence on such a case as this, where atrophy was sure to supervene, would be beneficial in retarding its onset, and limiting its degree. I therefore applied the current from six cells of Leclanché's battery for five minutes, reversing the poles before terminating. I also employed palpation in the hope that the clot might thereby be dislodged, but after the latter observed no change in the appearance of the vessels.

Next day vision was improved to $\frac{6}{36}$. The continuous current was again used as before.

On the following day, April 28, $V = \frac{6}{24}$, electricity again used. The perimeter chart taken showed the field of vision to be limited to a circle of less than 10 degrees radius round the fixation point.

On 29th $V = \frac{6}{18}$, there was some increase in the appearances of fatty degeneration of the retina, but the œdema was but slowly diminishing. Electricity again.

30th, same as on previous day.

May 1st (9th day), the first drawing and chart were taken; the œdema had somewhat subsided, and the sharp outline of its border near the macula was less exact. The periphery of the retina showed more stippling.

May 7 (15th day), atrophy was rapidly coming on, the disc was white, and the vessels much reduced in size. The field of vision had improved, especially at the outer side, where it now reached out to the 20 degree circle. $V = \frac{6}{12}$ and Jäg. 1, slowly. The battery had been used daily.

June 1st (the second drawing and chart were made). The battery had been used daily since last observation. The disc was perfectly white with well-defined edges. The vessels of both veins and arteries were very much reduced in size, but pulsation could be produced by pressure on the globe.

The region of the macula was marked by a peculiar goblet-

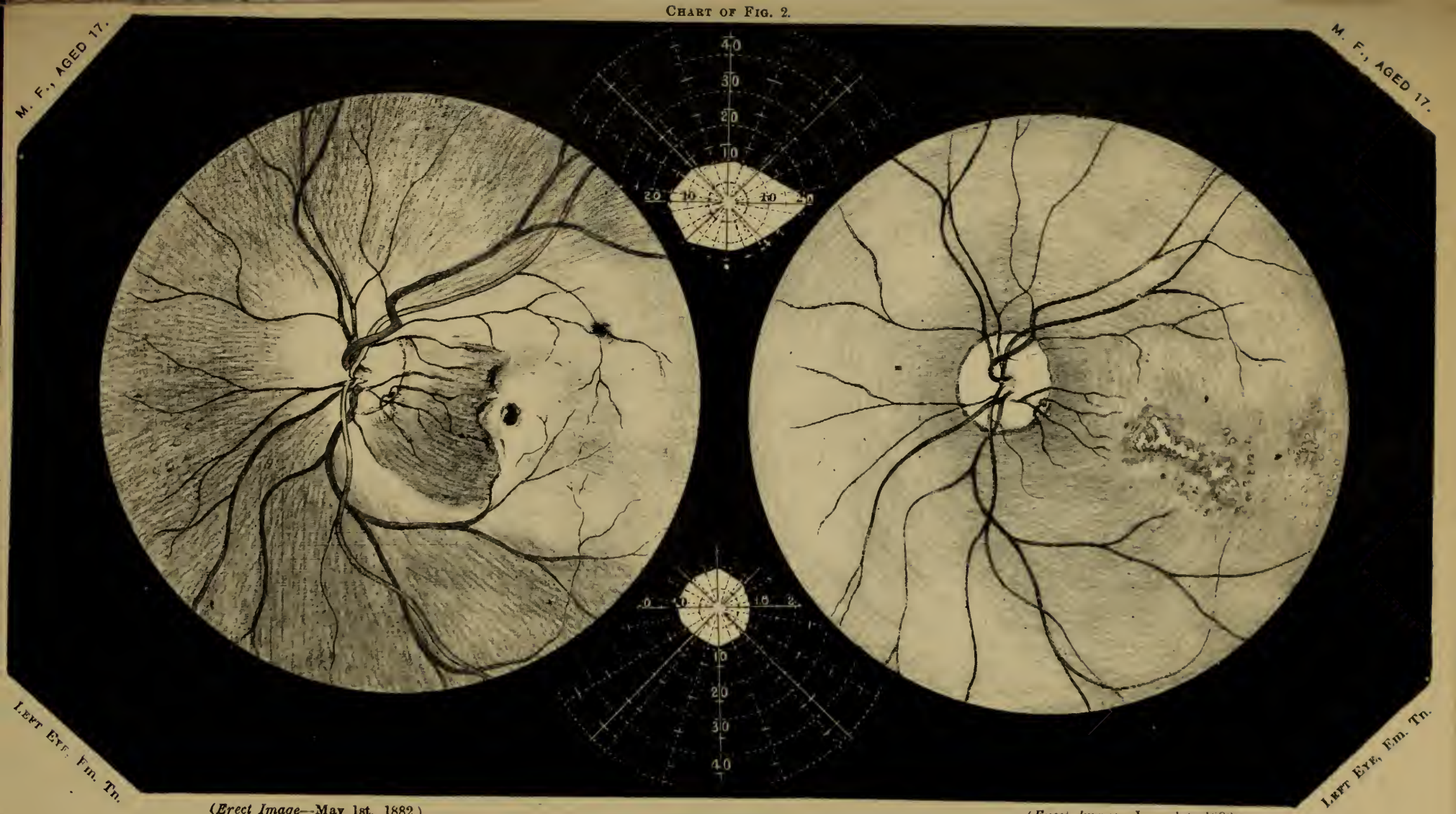
shaped patch of degeneration, much of its surface sparkling with brilliant yellow crystals. Some small bright crystals were also scattered irregularly about in the retina, from which the œdema had totally disappeared, T.n. V $= \frac{6}{12}$ and $\frac{6}{9}$, and Jäg. 1, slowly. At the outer side of the periphery, and to a less extent all round, there existed small round or oval white patches in the retina; they had well defined edges, and were nearly uniform in size, which was that of a small pin's head. They had more the appearance of exudations into than degeneration of the retina, but of their nature and cause I cannot offer any satisfactory account. The appearances were totally different from anything which I had before seen or read of.

The first question that suggests itself is, was the case really one of embolism of the central artery of the retina, or was it a thrombus in the vein, such as Michel* and Angelucci† have described, or apoplexy of the optic nerve as described by Magnus. If Angelucci's indications for differential diagnosis are to be taken as absolute, there would seem some probability that the case was one of thrombosis of the central retinal vein, but I think the weight of evidence still lies in favour of the obstruction being situated in the artery.

Had the treatment adopted in this case (viz., the continuous current) anything to do with the favourable result obtained? Many will say it had not, and perhaps they are right, but I cannot help thinking that if we bear in mind the slight improvement which had taken place since the accident, and the marked and steady improvement which followed the use of that agent; further, if we have found electricity so useful in other cases of atrophy of the optic nerve and retina, it must seem reasonable to ascribe some at least of the improvement to its employment here. Of course I do not mean to say that electricity will save a retina which is entirely and permanently deprived of blood by a complete block in the

* Graefe's Archiv. f. Ophthal., xxiv, 2.

† Annali di Ottalmologia, fasc ii, 1880.




(*Erect Image*—May 1st, 1882.)

Fig 1.

(*Erect Image*—June 1st 1882.)

Fig 2.

Drawn from life by ARTHUR H. BENSON.

main artery, but what I do say is that where you have a temporary blocking which soon gives way, but where there is strangulation of the retinal elements from the œdema, and where consequent atrophy is to be expected, not so much from loss of blood supply as from loss of functional activity, from pressure, &c., in such a case electricity may fairly be expected to benefit.

In the case I am here describing, only that portion of the retina recovered its function which had not been rendered œdematous, and which had probably not been deprived of its blood supply, but which nevertheless was struck blind simultaneously with the rest of the retina. The reason of its not becoming bloodless seems certain to be the existence of the cilio-retinal artery; but why it was in the first instance, and for so long rendered blind, is not so easily answered unless we assume the cause of the whole catastrophe to be a hæmorrhage into the optic nerve, which by pressure spoiled the conductivity of all the retinal fibre simultaneously, whilst in addition cutting off the blood supply from all that portion supplied by the central artery. As absorption occurred that portion whose circulation remained good recovered, whilst atrophy supervened elsewhere.

It was in saving this portion of the retina that I hold the electricity to have been helpful by supplying it with the stimulus it required for its nutrition during the absorption of the effusion, and perhaps also by hastening that process. It would probably have been ineffectual in restoring vision, but for the existence of the cilio-retinal vessel, and conversely it may be true that the cilio-retinal vessel might have been insufficient without the electricity.

The extreme sharpness of the line of demarcation between the œdematous and non-œdematous central patch, and its peculiar wavy line to the inner side, seems to require some anatomical explanation. It would seem as if the immediate region of the yellow spot was supplied by blood-vessels curving round from the disc, and that those vessels running directly towards the macula terminated before reaching its

immediate neighbourhood. It has been said with truth that pathology often makes more accurate dissections than the knife of the anatomist, and this may prove to be a case in point.

The appearance above mentioned would seem to depend more upon the separate vascular supply of the parts than on any arrangement of the retinal fibres, and, moreover, it appears that the cilio-retinal vessel does not really supply the region of the macula, as supposed by Mr. Nettleship in paper quoted above.

To recapitulate—the case seems remarkable—

(1.) For having occurred in a healthy person in whom no cause could be discovered.

(2.) For the existence of a cilio-retinal vessel.

(3.) For the extraordinary sharpness of the line of demarcation between the œdematous retina round the macula, and the normal patch to its inner side.

(4.) For the rapidity (less than three days) with which fatty changes occurred in the retina.

(5.) For the complete re-establishment of the circulation.

(6.) For the subsequent unusual appearances in the region of the yellow spot, and still more in the extreme periphery.

(7.) For the rapid restoration of sight whilst using electricity.

(8.) For the possible help the case may give in determining the blood supply in certain cases.

## VOLUNTARY NYSTAGMUS.

By Arthur H. Benson, M.B., F.R.C.S.I.,

*Assistant Surgeon to St. Mark's Ophthalmic Hospital, Dublin; Lecturer on Ophthalmic and Aural Surgery in the Ledwich School of Medicine, Dublin.*

The power of voluntarily producing rapid nystagmus is sufficiently rare to justify a brief note on the subject.

Mr. George Lawson has in the last number of these Reports mentioned the case of a gentleman who possessed this power to a remarkable degree. Shortly after the date of his paper, and before reading it, a young lady æt. 24, came to consult me about a curious twitching sensation which she at times experienced in her eyes.

She told me she could make her eyes "shake" whenever she wished. I asked her to do so, and she at once set both eyes into rapid lateral motion, the excursions to each side being very short, but succeeding each other with excessive rapidity, insomuch that the corneal outline and all the details of the globe were quite obscured. In a word, the eye shuddered. The motion, as Mr. Lawson says of his case, was really an exaggeration of what is seen in ordinary lateral nystagmus, but immeasurably more rapid. It was, however, completely under the control of the will, for she could start and stop the nystagmus at a second's notice, nor was there the slightest involuntary motion visible in the eyes. There was slight hypermetropia present.

## ON THE THEORY OF THE SO-CALLED KERATOSCOPY, AND ITS PRACTICAL APPLICATION.

By W. Charnley, M.A., M.D. (Cantab.)

I have attempted in this paper to give a brief account of the optical basis of the method of determining the refraction of the eye, which has been called keratoscopy or retinoscopy. The subject has already been treated by M. Parent of Paris, in two able articles which appeared in the "Recueil d'Ophthalmologie," in the numbers for February and July, 1880. I refer the reader to them for much valuable information. Mr. Morton in his work, "Refraction of the Eye," has given a sketch of the theory, and a very clear description of the practical working of the method.

As I find, however, that the subject is still unknown to not a few ophthalmic surgeons, I shall, for the sake of completeness, not only discuss the theory, but give what I hope may be a sufficient account of its application in practice; though in so doing I shall have to repeat much that has been already published. I may remark that neither of the terms hitherto used to describe the method appears quite satisfactory; keratoscopy suggesting a false theory, retinoscopy not being distinctive. Perhaps it would be best to call it "Cuignet's method," after the surgeon who first systematically described it.

What then is this method?

Suppose a patient placed in the ordinary position for ophthalmoscopic examination; the room darkened, the pupil dilated, and the gas-flame on the same side as the eye to be examined, and well to the rear of the head, so that no direct light from it reaches the patient's eye.

If we now place ourselves at about 48 inches from the patient, and reflect the light from the gas-flame into the eye in the direction of its visual axis, by means of a concave per-

forated mirror of about 9 inches focal length, we get the ordinary fundus-reflex.

Now suppose we give the mirror slight rotations about its vertical diameter, so that it "faces" in different directions, we then see something that has the appearance of a shadow moving horizontally across the illuminated area of the pupil: the edge of the shadow is seen to be vertical, and it moves (unless the observed eye be astigmatic, a subject to be fully discussed further on) either in the same direction as the mirror changes face, or in exactly the opposite direction.

If, for instance, rotating the mirror round its vertical diameter we turn it so that it faces to our right, the shadow moves horizontally either to the right or to the left. If it moves to the right, *i.e.*, in the direction in which the mirror changes face, the eye is Myopic; if it moves to the left, *i.e.*, in the opposite direction to that in which the mirror changes face, the eye is (generally) Hypermetropic. The observation we have just made is the germ of Cuignet's method. It is convenient to use the terms "with" and "against," in describing the movement of the shadow in reference to that of the mirror. For the future, then, we shall speak of the shadow moving "with" or "against" the mirror, according as it moves in the same or opposite direction to the change of face of the mirror.

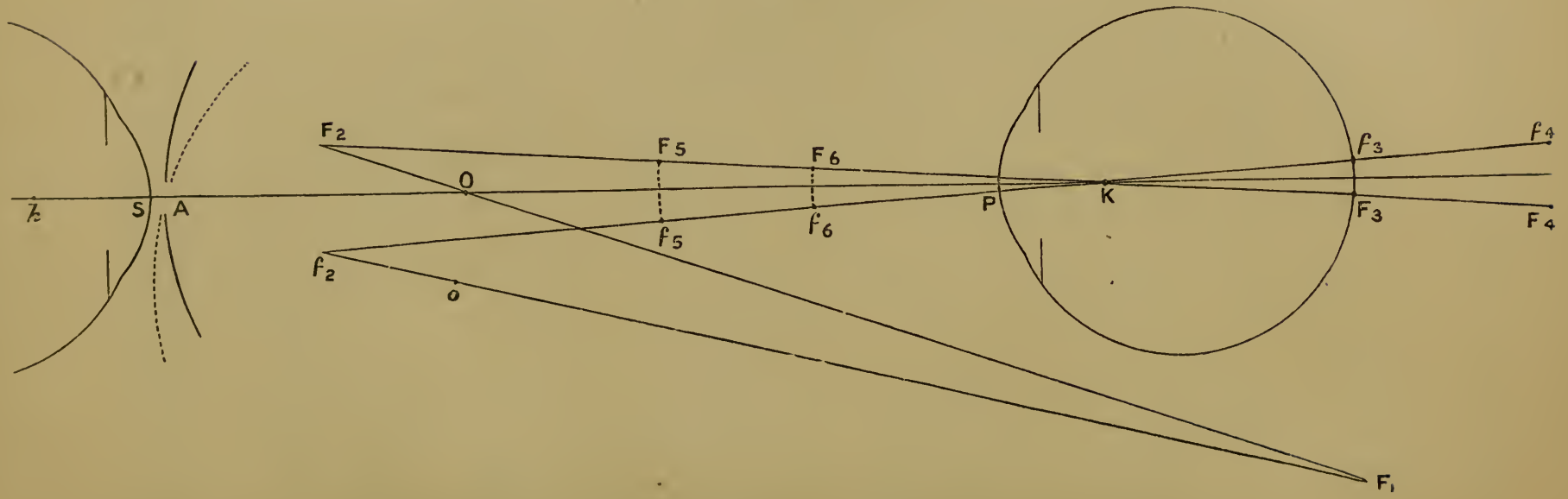
Now what is this shadow?

Suppose $F_1$ (Fig. 1) to be a luminous point, A the mirror held in front of the observer's eye $k$S, O the centre of the mirror, KP the observed eye, K being its nodal point, all these objects lying in the same horizontal plane.

An image of $F_1$ is formed by the mirror at $F_2$, $F_2$ being determined by drawing $F_1O$ and taking $F_2$ in $F_1O$ produced so that $F_1$, $F_2$ are conjugate foci. As $F_1$ is at a considerable distance from the mirror, $F_2$ will not be far from the principal focus of the mirror, so that $F_2$ is about 9 inches in front of A.

The rays of light diverging from $F_2$ strike the cornea of the observed eye and form an image at the point $F_3$ on the

FIG. 1.



retina, $F_3$ being the point in which $F_2K$ drawn through the nodal point K meets the retina.

Now $F_3$ being a point on the retina, an image will be formed, by the dioptric apparatus of the eye, of the image at $F_3$, the position of which will depend on the refractive condition of the eye. This image will in all cases be somewhere on the straight line $F_2KF_3$; if the eye be Hypermetropic it will lie behind its nodal point, at $F_4$, say; if the eye be Myopic it will lie in front of the nodal point at $F_5$, say; the distance $KF_4$, $KF_5$ depending on the amount of Ametropia, and being the less the greater the Ametropia is. If the eye be Emmetropic the image lies at an infinite distance.

To resume: the result of the reflection at the mirror A, and two successive refractions through the dioptric apparatus of the observed eye, is that an image of the luminous point $F_1$ is formed at $F_4$ in case of Hypermetropia; at $F_5$ in case of Myopia, and at infinity in case of Emmetropia.

This image will be a *point* only on the condition that $F_2$ lies at the far point of the eye KP; in that case rays diverging from $F_2$ will be accurately focussed on the retina at $F_3$, and the image here will be a *point*, and the image formed of this image will be a *point* at $F_2$.

If, however, the eye KP has not $F_2$ for its far point, instead of a *point* of light as the image at $F_3$ we have a *circle* of diffusion, and, consequently, the image at $F_4$ or $F_5$ is also a *circle*.

The observer then generally sees a *circle* of light at $F_4$ or $F_5$; but in the particular instance in which the eye KP has such an amount of Myopia that $F_2$ is its far point, he sees a *point* of light at $F_2$.

If now slight movements of rotation to the right round its vertical diameter be given to the mirror, so that the point A, remaining fixed, the centre O comes into the position *o*, and the mirror assumes the position shown by the dotted line, what becomes of the images?

In this case the new position of $F_2$, which we will call $f_2$, is determined as before by joining $F_1o$, and taking $f_2$ in

$F_1o$ produced, so that $F_1$ and $f_2$ are conjugate foci; $f_2$ is then clearly to the right of $F_2$, so that $F_2$ has moved "with" the mirror.

It is equally clear that $f_3$ is to the left of $F_3$, so that $F_3$ has moved "against" the mirror. What are the movements of the final images formed of the image at $F_3$ by the dioptric apparatus of the eye?

If the eye be Hypermetropic, the final position of the image is at $f_4$, so that the image has moved from $F_4$ to $f_4$, *i.e.*, "against" the mirror. If the eye be Myopic the image has moved from $F_5$ to $f_5$, *i.e.*, "with" the mirror.

*In Hypermetropia, then, the image of the luminous point appears to the observer to move "against;" in Myopia "with" the mirror.*

There are certain exceptions to these rules, however, which we may now consider. The observer is supposed to be placed at about 48 inches from the observed eye. Now suppose the latter to be Myopic; the final image is formed at $F_5$, and will be distinctly seen by the observer if he makes the effort of accommodation necessary to bring his far point to $F_5$. If the degree of Myopia be greater than 1 D., $F_5$ will be always far enough from the observer (supposed emmetropic and of good accommodation-power) for the image to be seen distinctly; as the Myopia diminishes, however, $F_5$ approaches S (the observer's cornea), and the image is no longer distinct. The indistinctness gets greater and greater, till at last the image is so blurred that no judgment can be formed of its movements.

If the Myopia still diminishes, $F_5$ falls to the left of S, and *convergent* rays fall on the cornea of the observer; these are at first so convergent that no distinct image can be formed on his retina; as $F_5$ passes still more to the left, they become less convergent, but now $F_5$ is to the left of $k$, the nodal point of the eye $k$S, so that the image of the image at $F_5$ formed on the retina is erect, instead of being inverted as it was when $F_5$ lay to the right of $k$. The image of $F_1$ then, formed on the observer's retina, has lost one inversion,

and as to each inversion of image corresponds an inversion of movement, the movement of the image in this case of low Myopia is opposite to that in ordinary cases of Myopia, where $F_5$ lies to the right of *k*. The image then moves "against" the mirror.

Hence, if the Myopia be less than 1 D., the movements of the image are either not seen at all (the image being so indistinct), or are "against" the mirror.

This particular degree of Myopia at which the inversion of movement takes place is determined solely by the distance at which the observer is from the observed eye.

Above, this distance has been taken as about 48 inches. If the distance were less, the degree of Myopia at which the inversion of movement took place would be greater. In fact it may be stated in general terms, that if the observer be near the far point of the observed eye, or within it, the image of $F_1$, formed at $F_5$, is either not seen distinctly at all, or moves "against" the mirror. But there are other exceptions to the general rule, which it will be interesting to examine.

Suppose the observer's eye *k*S armed with the mirror A approach the observed eye KP; then of course $F_2$ approaches P, and unless KP be very myopic the circle of diffusion at $F_3$ becomes so large that it ceases to be a distinct image, and its movements can be no longer distinguished.

As *k*S approaches still nearer KP, $F_2$ passes to the right of P, and rays enter the cornea of the observed eye *convergent*, but at first too convergent for a distinct image to be formed on the retina of KP.

When $F_2$ has passed to the right of K, a more distinct image is formed at $F_3$; but this is now erect with reference to the one at $F_2$; no inversion has taken place between these two points as is generally the case, and therefore the usual movements of the image are inverted; that is, in Hypermetropia the movement is "with" the mirror, in Myopia "against."

In the case of Hypermetropia the image is distinct, because the short hypermetropic eye is in a position to bring

to a focus on its retina the rays which now enter the cornea convergent.

In Myopia, on the contrary, the image is very indistinct, the rays falling convergent upon an eye which is adapted to bring to a focus on its retina only divergent rays; hence the movements of the image will not be so well seen as in Hypermetropia.

This movement "against" the mirror in Myopia is conditional on the far point of the Myopic eye lying to the right of the mirror, and not too near it for the observer to get a distinct image. If the Myopia be less than to satisfy this condition, the image is so indistinct that its movements cannot be appreciated, and becoming still less, the far point of the observed eye passes to the left of the nodal point of the observing eye, and the usual inversion of image produced by this eye does not take place. In this case two of the usual inversions of image are lost, one at the observed eye, one at the observing eye, and hence the image comes to move in the same way as in the ordinary case of Myopia, that is, "with" the mirror.

To sum up these movements of the image. In Hypermetropia the image always moves "against" the mirror, unless the distance of the observer from the observed eye be less than the focal length of the mirror, when the image, if distinctly seen at all, moves "with" the mirror.

In Myopia the image, as a rule, moves "with" the mirror; there are two exceptions:—

(1.) If the observer be nearer the eye than the far point of the Myopia, but not nearer than the focal length of the mirror, the image moves against the mirror.

(2.) If the observer be nearer the eye than the focal length of the mirror, but not nearer than the far point of the Myopia, the image moves "against" the mirror.

In the ordinary practice of the method, the observer being about 48 inches from the patient, the only exception that concerns us is (1) in Myopia. This we have already fully discussed.

So much for the *direction* of movement of the image; now as to the *extent* of movement.

Looking at Fig. 1 we see that corresponding to a movement of the centre of the mirror from O to *o*, the image moves in Hypermetropia through the distance $F_4f_4$, and in Myopia through the distance $F_5f_5$. Let us take the case of Myopia, and suppose the degree of it increased so that the far point of the eye KP lies nearer P, say, at $F_6$; on rotating the mirror through the same angle as before so that O passes to *o*, $F_6$ will pass to $f_6$. Now $F_6f_6$ is clearly less than $F_5f_5$, hence, as the Myopia increases, the extent of movement of the image diminishes.

Similarly, it may be shown that as the Hypermetropia increases the extent of movement of the image diminishes.

Further, as to the *brightness* of the image. We have seen above that only in the case of the far point of the eye KP coinciding with $F_2$ (M = 1 D. about), is the image of $F_1$ formed on the retina of the observed eye as a point; in the other cases the image is a circle of diffusion, the diameter of which increases as the refractive condition of the eye departs from this amount of Myopia in either direction, that is, either towards higher Myopia, or Emmetropia to Hypermetropia.

Now, a Myopia of 1 D. does not differ much from Emmetropia; so we may say roughly that the circle of diffusion on the retina of KP increases with the Ametropia. Since the same amount of light is in all cases concentrated into this illuminated circle, it follows that any point of this circle will be less illuminated the higher the Ametropia.

Moreover, the rays proceeding from a point of this circle of diffusion converge to or diverge from a point the nearer K, the greater the Ametropia, and are therefore dispersed over a greater angle, so that fewer rays enter an aperture of given size, like the pupil of the observer, the greater the Ametropia.

Hence the image of $F_1$ seen by the eye *k*S is less illuminated in the higher degrees of Ametropia for two reasons:—

(1.) Because the illumination at any point of the image formed on the retina of the eye KP is less.

(2.) Because this comparatively deficient illumination is itself diminished by the greater dispersion of the rays in their passage through the dioptric apparatus of this eye.

We see then, (*a*) that the image of the point $F_1$, formed by successive reflection at the mirror A, refraction into the eye KP, and refraction out of this eye moves (with certain exceptions) "with" the mirror in Myopia, "against" it in Hypermetropia; (*b*) that the greater the Ametropia, the less the extent of movement, and (*c*) the greater the Ametropia the less the illumination of the image.

What as to Emmetropia?

This may be considered as Myopia of excessively low degree, the far point being, indeed, at infinity. Hence, it comes into the category of the exceptional cases of Myopia, and the image moves "against" the mirror; and the degree of Ametropia being excessively small, the extent of movement is excessively large, and the illumination of the image at its maximum.

If instead of a luminous point at $F_1$, we have a flame, which may be considered as a congeries of luminous points, all that has been said above equally applies.

In this case we have formed on the retina of the eye KP an image more or less precise, according to the refractive condition of this eye; if it be Myopic to the extent of 1 D., an image is formed on the retina with sharply-defined edges, and exactly reproducing the shape (generally with our burners rectangular) of the flame at $F_1$. As the eye departs from this condition of slight Myopia, the retinal image will be composed of a collection of circles of diffusion, having as centres points on the retina corresponding to the points of light which may be supposed to make up the flame at $F_1$. This image will be then roughly circular. We have in this case formed on the retina a roughly circular illuminated area, in place of the one exactly circular, when we considered the source of light as a luminous point.

All that has been proved of the image in the one case is true of the image in the other, and the same statements may

be made as to the movements, &c., of this roughly circular image as on p. 352.

It remains to consider the *distinctness of the edge of the image*. M. Parent, in the articles before referred to, states that this distinctness is proportional to the degree of Ametropia, being the greater the higher that degree is, and even uses it as a rough test of the amount of Ametropia. I must confess my experience does not agree with his. I am, indeed, accustomed to use the intensity of fundus-reflex, and the extent of movement of the image, as an approximative test of the refraction, but I have not been able distinctly to see the variations of distinctness of edge which M. Parent describes, and the theory of the formation of the image would lead me to expect this increasing distinctness with increasing Ametropia only in the case of Hypermetropia, and not to be well-marked even then.

It may be worth while to investigate this more fully.

It has been shown above that in Myopia of about 1 D. we have an exact image of the source of light produced on the retina of the observed eye; in other states of refraction, the edges of this image are more or less blurred and indistinct; a measure of this indistinctness is the diameter of the circles of diffusion, which we have already seen are the greater, the greater the departure from a Myopia of 1 D.

Hence the greater the departure from this condition of refraction, the more indistinct the image formed on the retina of the observed eye.

It might be supposed then that the definition of the edge of the image seen by the observer would be the less the greater the departure from Myopia = 1 D. We must remember, however, that what the observer sees is not the image formed on the retina of the eye KP, but the image formed of this image at the far point of this eye. It is the definition of the edge of this latter image that we are concerned with. A measure of this definition is the diameter of the circles of diffusion in this image. This we proceed to investigate.

We take Donders' reduced eye for the purpose of our calcu-

lations, and suppose the different degrees of Ametropia produced by changes in the length of its axis.

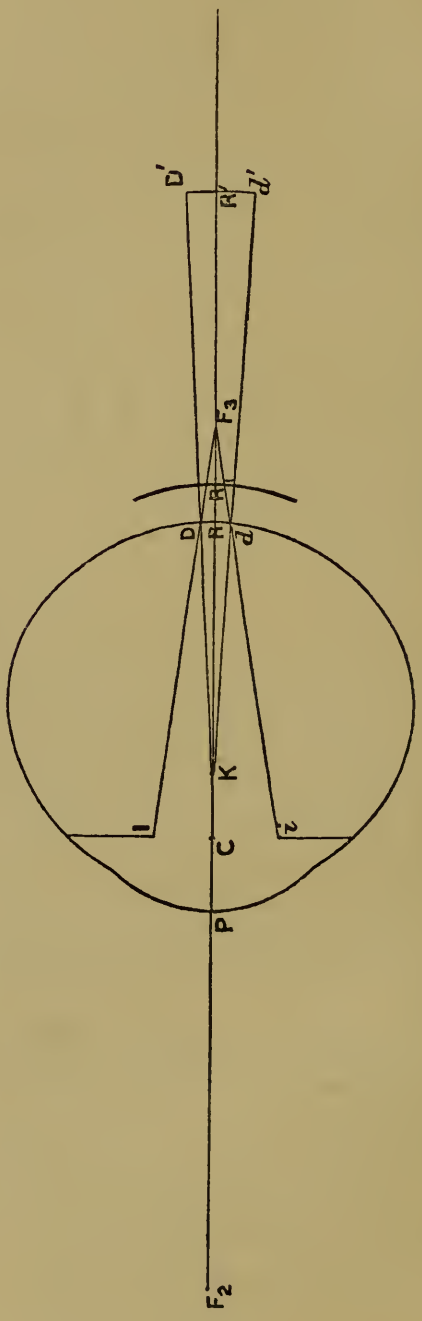



Fig. 2.

Suppose the eye KP hypermetropic to the extent of $n$D.; let R be its retina, $R_1$ the retina of the emmetropic eye, $F_3$

the point to which rays diverging from $F_2$ (supposed on the axis of the eye) converge after refraction through the eye; $Ii$ the pupil, C its centre; $l$ the distance from the plane of the pupil to the retina of the emmetropic eye, that is $CR_1$; $Dd$ is then the circle of diffusion; its image is at $D'd'$. We have then to find $D'd'$.

We suppose all lengths expressed in millimeters.

From similar triangles—

$$\frac{Dd}{Ii} = \frac{F_3R}{F_3C}, \text{ and } \frac{D'd'}{Dd} = \frac{KR'}{KR};$$

$$\therefore \frac{D'd'}{Ii} = \frac{F_3R}{F_3C} \times \frac{KR'}{KR}.$$

Now $F_3R = F_3R_1 + R_1R$,

and $F_3R_1 = \frac{3}{10}$ (nearly), $F_3$ being conjugate to $F_2$, which is about 1 meter in front of the eye, and $R_1R = \frac{3n}{10}$*

$$\therefore F_3R = \frac{3}{10}(1 + n),$$

$$\text{and } F_3C = F_3R_1 + R_1C = \frac{3}{10} + l;$$

$$\therefore D'd' = Ii \times \frac{\frac{3}{10}(1 + n)}{\frac{3}{10} + l} \times \frac{KR'}{KR}.$$

Now R, R′ being conjugate foci with respect to the refracting surface of the reduced eye,

$$\frac{\mu}{PR'} - \frac{1}{PR} = \frac{\mu - 1}{r},$$

$$\text{where } \mu = \frac{3}{4},\ r = 5.$$

$$\text{And } PR' = 5 + KR',\ PR = 5 + KR;$$

$$\text{moreover } KR = KR_1 - RR_1 = 15 - \frac{3n}{10};$$

therefore the above equation becomes—

$$\frac{3}{4(5 + KR')} - \frac{1}{20 - \frac{3n}{10}} = -\frac{1}{20},$$

* Cf. Donders. "Accommodation and Refraction." New Syd. Soc. Transl., Lond., 1864, p. 178.

$$\text{whence } KR' = \frac{1000 - 20n}{n},$$

$$\text{and } \frac{KR'}{KR} = \frac{200}{3n};$$

$$\therefore D'd' = Ii \times \frac{\frac{3}{10}(1 + n)}{\frac{3}{10} + l} \times \frac{200}{3n}$$

$$= Ii \times \frac{200}{3 + 10l}\left(1 + \frac{1}{n}\right).$$

. ·. as $n$ increases $D'd'$ diminishes; that is, the diameter of the circle of diffusion in the image seen by the observer is less the greater the Hypermetropia. As the definition of the edge of this image is greater the smaller the circle of diffusion, we obtain the result, that the definition of the edge of the image seen by the observer is greater the greater the degree of Hypermetropia in the observed eye.

In a similar way it may be shown that in Myopia the diameter of the circle of diffusion $= Ii \times \frac{200}{3 + 10l}\left(1 - \frac{1}{n}\right)$.

This is least when $n = 1$, being then 0. In other words, the definition of the edge of the margin is greatest with Myopia = 1 D., the image then being perfectly distinct. This is evident from other considerations adduced above.

As the degree of Myopia increases or diminishes from 1 D., the image grows less distinct.

These variations of distinctness of image are so difficult to appreciate, that I cannot say certainly whether the above calculation is confirmed by observation or not. I find it best, if I am called upon to make a rough estimate of the refraction, without using correcting glasses, to depend not on the distinctness of the edge of the image, but on the brightness of fundus-reflex, and the rapidity of movement of the image.

We are now in a position to answer the question (proposed on p. 345)—What is this shadow?

It is simply the edge of the image of the gas-flame, formed by one reflection at the mirror, and two refractions through the media of the patient's eye.

The movements of the shadow are the movements of the

edge of the image referred to the margins of the pupil of this eye. The edge of the image is more or less circular, but as only a small part of it is generally in the area of the pupil, it will appear as a shadow with a nearly straight edge perpendicular to the direction of movement.

I recapitulate what has been proved about this shadow—

(1.) It moves as a rule "with" the mirror in Myopia; "against" it in Hypermetropia.*

(2.) The extent of movement is inversely proportional to the amount of Ametropia.

(3.) In Myopia the shadow is most distinct in a Myopia of 1 D., and diminishes in distinctness as it varies from this amount.

(4.) In Hypermetropia the shadow is more distinct the greater the Hypermetropia. [These variations of distinctness are however not easily seen in practice.]

To these may be added—

(5.) The fundus-reflex is less intense the greater the Ametropia.

The method of examination has been already briefly described. It is necessary to add a few words to that description. The side of the patient's head should be shaded from the direct light of the gas-flame by a screen; the observer, if not emmetropic, must correct his Ametropia, and the light should be thrown into the patient's eye nearly in the direction of its visual axis. If the pupil be under atropine, it may be thrown in exactly along that axis, the patient being directed to look at the mirror; if atropine have not been used, the pupil contracts so much when the light is thrown in directly, that the shadow cannot be seen easily; we must, therefore, so direct the light, that it falls on the retina somewhere near the optic disc.

We supposed above that the rotations were made around the vertical diameter of the mirror; but any other diameter, horizontal or oblique, does of course just as well. The direc-

* If the mirror be plane, or convex, these movements are reversed.

tion in which the mirror is being rotated is best appreciated by noticing the movement of the circular patch of light on the patient's eye and brow; this is the section made by the patient's face of the bundle of rays diverging from the image formed by the mirror at $F_2$; it moves "with" the mirror of course; its extent of movement is the best standard to refer to when we are estimating the extent of movement of the shadow.

Suppose then the patient and surgeon arranged as just directed, the light thrown into the patient's eye, and the movement of shadows observed, how do we diagnose the kind and amount of Ametropia? The following paragraphs will explain:—

If with usual gas-light the fundus-reflex is very *dull*, we infer a high degree of Ametropia; this is confirmed by the smallness of the extent of the movement of the shadow. If the shadow moves "with" the mirror, it is certainly a case of Myopia. If the shadow moves "against" the mirror it is a case of high Hypermetropia. (N.B. It is supposed that there are no pathological changes in the media of the eye to account for the dull fundus-reflex.)

If the fundus-reflex be bright, we infer a low degree of Ametropia; this is confirmed by the wide extent of movement of the shadow. If the shadow moves "with" the mirror, it is certainly a case of Myopia. If the shadow moves "against" the mirror, we cannot certainly say without further examination whether we have to deal with low Hypermetropia, Emmetropia, or low Myopia, as all these conditions give a shadow moving "against."

If the fundus-reflex be bright, but no distinct shadow can be got, but only vague indefinite obscurities, the movement of which cannot be appreciated, it is a case of very low Myopia, the far point of the observed eye falling near the nodal point of the observer's eye.

It is this condition we endeavour to bring the eye to, in estimating its refractive condition by placing glasses in front of it. It is convenient to speak of the eye in this state (that is, when with bright fundus-reflex no distinct shadow is seen)

as "corrected." It must be remembered, however, that the eye so "corrected" is not emmetropic, as the term might imply, but has a low Myopia, say about 0·50 D.

How we bring about this state of correction will be best seen from an example.

Suppose the shadow moves "with" slowly, and the fundus-reflex is dull, we infer a high degree of Myopia.

Let us put up then a pretty high negative lens before the patient's eye in the spectacle-frame, say — 8 D.

If the shadow still moves "with," we know that the eye *plus* the lens is still myopic. Let us then gradually increase the strength of the concave lens till we reach the stage where we get no distinct shadow at all, that is, the stage where the eye is "corrected." We now know that the combination of the patient's eye and the negative lens is now myopic about ·50 D.

Suppose — 9 D. the lens that brings the eye to this state; then about 9·50 D. would make the combination emmetropic; So 9·50 D. is the full measure of the Myopia. And in all cases the Myopia is about ·50 D. greater than the negative lens which "corrects" the eye.

If — 8 D, the lens originally placed in the spectacle-frame, makes the shadow move "against," we must gradually *diminish* the strength of the negative lens till we reach the "corrected" stage.

It is easily seen that the degree of Hypermetropia is about ·50 D. less than the positive lens which "corrects" the eye.

We now see how to discriminate the three cases mentioned above, viz., low Hypermetropia, Emmetropia, and very ow Myopia, in all of which the shadow moves "against."

A negative lens of ·50 D. would still leave the shadow moving "against" in the first case, would bring the Emmetropia to the corrected stage, where no distinct shadow is seen at all, and in the low Myopia convert the movement "against," into a movement "with."

I find it most convenient in practice to bring the patient's eye to the "corrected" stage, and make afterwards what

slight alterations in the glass may be necessary, in testing with Snellen's types at 20 feet. We might, instead of aiming at this stage of "correction," estimate the Myopia by the weakest concave lens which made the shadow move "against," and the Hypermetropia by the strongest convex lens which still left it moving "against."

These lenses would probably very nearly neutralise the Ametropia.

Hitherto we have supposed the refraction the same in all the meridians of the eye; that there was no Astigmatism.

We must now consider what happens in Astigmatism.

Here rays of light after passing through the dioptric apparatus of the eye no longer converge to a point; they converge to two (approximately) straight lines that lie at the anterior and posterior boundaries of the focal interval of Sturm.*

As before, let us suppose for a moment that the source of light is a luminous point; the image of this formed by the mirror at $F_2$ (Fig. 1) is also a luminous point, but the image formed on the retina of the eye KP is no longer a circle; it is in general an oval—an oval of diffusion it might be called. The shape and dimensions of this oval vary with the position of the retina, that is, with the refraction of the eye. For a discussion of these various shapes, &c., we must refer to Donders (*loc. cit.*). The following facts are there shown:—

If VPV′ (Fig. 3) be the meridian of greatest curvature, supposed for simplicity's sake to be vertical, and in the plane of the paper, the meridian of least curvature, HPH′, therefore perpendicular to the plane of the paper, $v$ the focus to which rays passing from the luminous point, $F_2$, through the meridian, VPV′ converge, $h$ the point to which rays from $F_2$ converge in the meridian HPH′; then the retina placed at $v$ receives a linear image of $F_2$ perpendicular to the plane of the paper; to the left of $v$ the image is an oblate oval; as the retina changes position from $v$ to the right, the image of

* Donders. *Op. cit.*, p. 457, &c.

$F_2$ formed on it is, successively, an oblate oval between *v* and some point *o* between *v* and *h*, where it is a circle; a prolate oval between *o* and *h*; at *h* it becomes a straight line in the plane of the paper perpendicular to the axis; beyond *h* it is still a prolate oval.

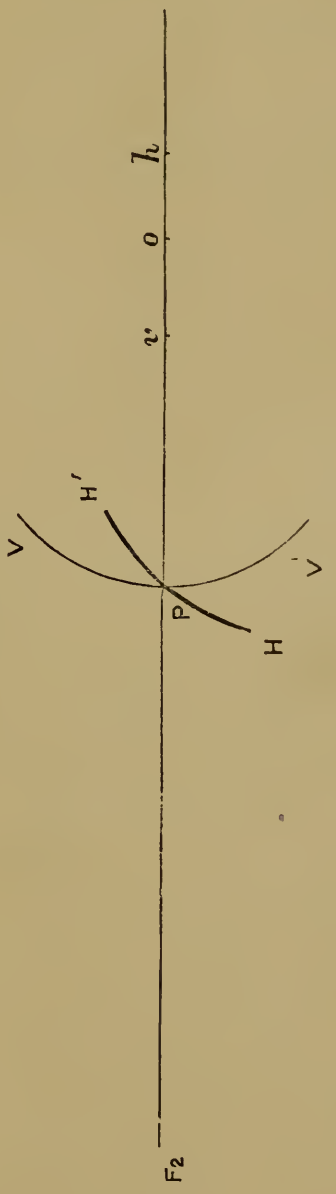


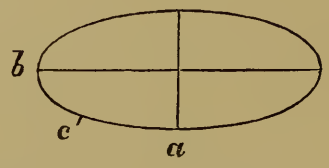

Fig. 3.

It may be shown that when the source of light is no longer a luminous point, but a flame of finite magnitude, the

image on the retina of the eye takes the shape of the area of diffusion, and is therefore generally an oval; at $v$ this oval becomes a straight line perpendicular to the plane of the paper; at $h$ a straight line in the plane of the paper perpendicular to the axis. What the eye of the observer sees is not of course this image, but the image formed of it by the dioptric apparatus of the eye. Where, then, will the image be formed ?

Suppose the retina of the eye is situated a little to the right of the point $v$, then the retinal image is an oblate oval (Fig. 4).

Fig. 4.




Let us consider where the image of the horizontal edge of this oval will be formed. As before, rays proceeding from any point $a$ on this edge will, after refraction through the unequally curved meridian of the eye, converge to or diverge from, not a point, but two straight lines (the focal lines). The image of the edge will be referred to the position where it is most distinctly seen, that is, to the position where the images of adjacent points on the edge are drawn out into ovals whose long axes are horizontal, and therefore overlap and so cause the greatest condensation of rays. Now this position is at the far point of the vertical meridian, where indeed the images of $a$ and adjacent points are ovals so elongated that they may be considered horizontal straight lines. The position of the image of the *horizontal* edge of the retinal image depends then on the refraction of the *vertical* meridian of the eye, and is in front of, behind the eye, or at infinity, according as the eye is myopic, hypermetropic, or emmetropic. The horizontal edge, then, of the retinal image and the vertical meridian in the astigmatic eye, stand then in the same relation as the whole image and any meridian in the spherical eye.

Hence the movements of the image of the edge (itself a horizontal edge) will follow the rules already deduced, that is if the vertical meridian be Myopic the movement will be "with" the mirror; if Hypermetropic, generally "against."

Similarly the movements of the image of the *vertical* edge of the oval retinal image depend on the refractive condition of the *horizontal* meridian of the eye.

We have then as a criterion of the refraction of either of the principal meridians of the eye a shadow moving parallel to that meridian, and whose edge is perpendicular to that meridian.

Of a part of the edge of the oval near *c* intermediate to *a* and *b*, we get no distinct image formed, because the images of adjacent points are ovals whose long axes are either horizontal or vertical, whilst these adjacent points are arranged neither horizontally nor vertically with respect to one another. Hence we have only two distinct shadows; these are perpendicular to the principal meridians of refraction, that is, horizontal and vertical.

We have supposed principal meridians to be horizontal and vertical for the sake of simplicity. Frequently, however, they are oblique. In this case the distinct shadows, which are always perpendicular to the principal meridians, will also be oblique, and, further, their movements will be oblique.

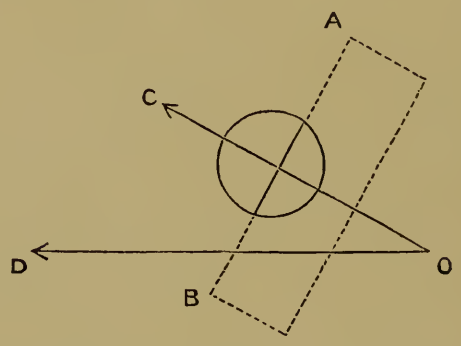
This obliquity of movement of the shadows depends on an optical illusion, and is independent of the direction in which the mirror is rotated.

Suppose, for instance, the principal meridians are inclined at an angle of 45° to the horizontal line, then the form of the retinal image is an oval with its axes at the same angle to the horizon, and therefore the two distinct shadows are also inclined at 45° to the horizon.

Now let the mirror be rotated horizontally to the right (that is, let the illuminated circle on the patient's face be made to pass horizontally to the right). Then the oval retinal image moves horizontally to the left, and the shadows, which are the images of the long and short edges of the oval, also move horizontally; whether they move to the right or to the

left depends on the condition of refraction of the meridians to which they are respectively perpendicular. But although the *absolute* movement of each shadow is horizontal, its apparent movement is always perpendicular to its own edge. This is a consequence of the optical illusion just referred to, which may be thus illustrated. Suppose we have a circular aperture, and behind it a piece of card with a straight edge, AB is passed without our knowing in what direction it is being passed.

Fig. 5.




Then the *apparent* movement is always perpendicular to its edge; that is, in the direction of the arrow C. If, for instance, the card is moved horizontally in the direction of the arrow D, it will still appear to move in the direction of C. The apparent rate of movement is the less than the real, the greater the angle between the directions of the arrows C and D. In fact if these directions meet in O, we have rate in direction OC = rate in direction OD × cos COD.

Now this is an exact reproduction of what takes place in the astigmatic eye, the pupil representing the circular aperture, and the edge of the shadow the edge of the card.

We get in this case, then, with horizontal rotations of the mirror (rotations around a vertical axis), an oblique shadow moving obliquely. This shows at once that there is Astigmatism, and shows, further, that the principal meridians are parallel and perpendicular to the shadow.

If we now rotate the mirror so that the illuminated circle on the patient's face moves in the same direction as the

shadow, we find at once that the latter moves more quickly, it being in fact now its whole motion that we see, and not merely a resolved part of it, as was the case when the rotation of the mirror and the movement of the shadow was not in the same direction.

It may be asked which of the two distinct shadows corresponding to the principal meridians do we see when the movement of the mirror is not in the direction of either of them? Suppose, for instance, the principal meridians to be inclined at 45° to the horizon, and the mirror to be rotated horizontally; since this direction of movement is equally inclined to the two principal meridians, it seems that either shadow has the same chance of being seen. The fact is, it is sometimes one, sometimes the other; but generally the shadow corresponding to the meridian of greatest Ametropia is predominant; this finds an explanation in the fact that this shadow is the most intense. *Cæteris paribus*, that shadow is most likely to be seen with whose chief meridian the movement of the mirror most nearly coincides.

Let, for example, the meridian inclined 20° to the vertical have H = 5 D., the meridian inclined 110° to the vertical have H = 3 D. The shadow corresponding to the first of these has the greatest intensity; but with horizontal rotations of the mirror, I should expect the other one to be predominant, because the inclination of the absolute to the apparent movement in the first case is 70°, in the second only 20°.

If conditions are nearly equally favourable to the production of each shadow, we do not get anything definite till we begin to correct by lenses; as then one meridian approaches Emmetropia or, more strictly speaking, a condition of M = ·50 D., its shadow becomes very faint, and then the shadow corresponding in the other meridian stands out boldly. Hence simple Astigmatism is easily detected. If the principal meridians be vertical and horizontal, horizontal or vertical movements of the mirror (which we generally begin with) do not show this incongruity of movement, and therefore do not reveal the Astigmatism. But if we make the movements of

the mirror oblique, the shadows appear horizontal or vertical, and betray the Astigmatism, and the direction of the principal meridians. The obliquity of the meridians is to be estimated by the observer's eye; of course it cannot be done exactly, but we can get within 10° probably.

We see, then, how Cuignet's method enables us to detect the two principal meridians of the eye under examination.

Having determined these we proceed to examine the refraction of one of them, transferring the movement of the mirror to its direction for that purpose. We have then as a criterion of the refraction of this meridian a shadow moving parallel to it, and with its edge perpendicular to it. All that has been proved of the shadow in any meridian in the non-astigmatic eye is true of this one meridian and the shadow perpendicular to it, and the conclusions stated on p. 357 may be repeated as applying to this particular meridian.

The movement of the shadow will then show whether it is Myopic or Hypermetropic; having ascertained this, we proceed to put up the negative or positive lenses respectively till we reach to the stage of "correction," where we get no well-defined shadow. We note the lens required, and then proceed to correct the meridian at right angles to this, making now the illuminated patch on the patient's face move in the direction of this meridian. We may now put up a combination of sphere and cylinder which corrects both principal meridians, and should then get no distinct shadows in any direction.

We now proceed to find what the patient can read at 20 inches with the correcting glass, and seeing that the stage of "correction" is one of about ·50 D. of Myopia, we should expect to have to diminish the positive or increase the negative sphere by this amount before we get the best spherical lens.

Frequently this is so.

In any case we try slight changes in the spherical lenses till we get the best vision at 20 inches, then slight changes in the strength of the cylindrical lens, and the direction of its axis.

In discussing the theory of this method of examination, I have supposed the observer to be at about 48 inches from the observed eye. This distance was suggested as convenient by M. Parent in the articles before referred to. In practice, I find a distance of about 36 inches more convenient; the patient is more within one's reach, and the correcting lenses can be changed without the examiner's getting up.

This shorter distance has the disadvantage of increasing the amount of Myopia corresponding to the stage of "correction." This is not a great drawback.

At this distance with the usual concave mirror the illuminated circle on the patient's face has a diameter of about 2 inches, or the interval between the eyes. The size of this circle will roughly indicate whether the observer is at the right distance. The surgeon should get into the habit of always placing himself at the same distance, whether it be 36 or 48 inches; and the same intensity of light should always be employed.

At the risk of reiterating needlessly, I here give a *résumé* of the procedure I generally adopt, and the conclusions that can be drawn at each stage.

The usual precautions as to light, position of the patient, &c., being observed, I direct the light into the patient's eye.

If the fundus-reflex is dull, the Ametropia is high; if bright, low.

I now make small rapid rotations of the mirror so that the circle of light on the patient's face travels horizontally and vertically in succession.

If the shadow travels horizontally and vertically respectively, there is not much Astigmatism with principal axes oblique. I now make oblique rotations of the mirror in one or two directions; if the shadow still moves parallel with the rotations, there is not much Astigmatism at all. The direction of movement of the shadow "with" or "against" has indicated Myopia or Hypermetropia.

I now proceed to "correct" the eye.

The patient is fitted with a spectacle-frame having a

double clip before each eye, and I place in the frame before the eye a positive or negative lens, according as the rotations of the mirror have shown the eye to be myopic or hypermetropic. The strength of the lens selected depends upon the judgment I have formed of the amount of Ametropia, from the intensity of the fundus-reflex, and the extent of movement of the shadow. With some experience, an approximation to the correcting lens may be made at the first trial. I now see how the shadows move in the eye armed with the lens placed in the frame. If they still move in the same sense as before, I know the eye is still under-corrected, and increase the strength of the lens in the frame, either changing it for one stronger by 1 D., or placing before it in the second clip of the frame an additional lens of 1 D. of the same sign. I test the effect of this new (combined) lens by horizontal and vertical rotations of the mirror, as before. If necessary, I increase the strength of the lens still more, by successive increments of 1 D., until either I reach the stage of correction, when no distinct shadows are seen; or the shadows move in a direction opposite to that in which they originally moved.

In the latter case I know that the eye is now over-corrected, and reduce the strength of the lens by 0·50 D. This probably "corrects" the eye. If it does not, I accept it as an approximation, knowing that it differs from the "correcting" lens by certainly less than 0·50 D., and having in view that the final test is with the types at 20 feet.

As the stage of correction is being approached, the characteristic appearances of low degrees of Ametropia are noticed, viz., bright fundus-reflex, and increased extent of movement of the shadow; not unfrequently, too, Astigmatism that had previously escaped notice, manifests itself by the appearance of an oblique shadow.

If this is not the case, and the horizontally- and vertically-moving shadows produced by horizontal and vertical rotations of the mirror disappear with the same lens, I know there is no Astigmatism.

I have now reached the stage of "correction," and am prepared to find that the "correcting" lens will have to be slightly diminished if convex, increased if concave to give the best vision at 20 feet.

If, with the horizontal and vertical rotations of the mirror, one of the horizontally- and vertically-moving shadows has disappeared before the other, or come to move in the reverse direction, I know there is Astigmatism with the principal meridians, horizontal and vertical. I proceed then to "correct" each of these meridians separately by making the rotations of the mirror in the direction of the meridian under examination, and finding what spherical lens makes the shadow moving in the direction of this meridian disappear. I put up then a sphero-cylindrical combination which "corrects" both axes, and then expect to get no shadow in any direction. I am prepared to find that this combination requires slight spherical alteration to give the best vision at 20 feet.

If at any stage of the examination Astigmatism with principal meridians oblique has manifested itself by obliquely moving shadows, though the rotations of the mirror are horizontal and vertical, I note the directions of the principal meridians, transfer the rotations of the mirror to the direction of each meridian in turn, and correct them precisely in the same way as if they were horizontal and vertical.

A few examples taken from actual practice may be useful in explaining the procedure.

Case 1. Myopia. No atropine.

Right eye. Dull fundus-reflex. Shadow moves slowly "with" in all meridians; — 9 D. "corrects" the eye; with — 10 D. letters of $\frac{20}{30}$ are read.

This follows the rule that the negative glass giving the best vision at 20 feet is rather stronger than the one that "corrects" the Myopia.

Case 2. Hypermetropia.

Right eye. No atropine. Moderate fundus-reflex. Shadow

moves "against;" + 5·50 D. corrects all meridians; with + 5 D. $\frac{20}{20}$ is read.

Case 3. Hypermetropic Astigmatism. Aphakia.

Dull fundus-reflex. Shadow moves slowly against; + 11 D. "corrects" the horizontal meridian; + 10 D. "corrects" the vertical meridian; with $\frac{+ 10 \text{ D}}{- 1 \text{ D cyl.}}$ axis horizontal $\frac{20}{20}$ read.

Here, again, the correcting convex glass has to be diminished.

Case 4. Hypermetropic Astigmatism.

Right eye. Dull fundus-reflex. Shadow moves "against." With + 8 D. a well-defined shadow moving "with" the mirror in a meridian 45° up and out; no shadow seen in meridian perpendicular to this: that is + 8 D. "corrects" this latter meridian. With + 6 D. no shadow in the meridian inclined 45° up and out; in the meridian perpendicular to this a faint shadow moving "against," that is + 6 D. "corrects" the former meridian; $\frac{+ 6 \text{ D}}{+ 1{\cdot}50 \text{ D cyl.}}$ axis 45° up and out "corrects" all the meridians, so that no shadow is seen in any direction. The lens that gives the best vision at 20 feet is $\frac{+ 5{\cdot}50 \text{ D}}{+ 1{\cdot}50 \text{ D cyl.}}$ axis 50° up and out, and with this $\frac{20}{30}$ is read, V without the cylinder being only $\frac{20}{100}$.

I have chosen these cases to illustrate the chief anomalies of refraction.

The method is much simpler in practice than long descriptions of it on paper would suggest. A few minutes' practical instruction are sufficient to teach the necessary manipulation, and the observation of the direction movement of the shadows in simple cases.

Some experience is necessary for the ready appreciation of what I have called the "corrected" stage, and still more for the estimation of Astigmatism, especially when the chief meridians are oblique.

As a qualitative test, it must be admitted that the method is unrivalled; a glance at the eye suffices to show whether it is Myopic or Hypermetropic, and generally whether it is Astigmatic or not.

There may be a difference of opinion as to its merits as a quantitative test. It has the immense advantage of all objective methods, that it does not leave us dependent on the intelligence of patients.

I think it is superior to other objective methods, especially in the ease and certainty with which it enables us to determine the principal meridians of an Astigmatic eye.

For my own part, I always employ it when I suspect Astigmatism, and in children for the correction of even a spherical anomaly. It has been extensively tried in England, since its first introduction here, and I know of scarcely an instance in which it has been fairly tested, and failed to please. The surgeon who has much to do with refraction must have no doubt many weapons at his command; I think he will do well to add to his list, if he have not already done so, this method of Cuignet.

## ON THE SIZE AND POSITION OF THE CRYSTALLINE LENS IN GLAUCOMA.

By W. A. Brailey, M.A., M.D. (Cantab).

*Curator of the Museum, Roy. Lond. Ophth. Hosp.*

During the last two years I have made observations as to the size of the lens in eyes excised at Moorfields Hospital.

At first I contented myself with recording cases of glaucoma, in which the lens seemed unusually large. Then I made measurements in every case, whether the lens was cataractous or not.

The general result was to show that the average size of the lens in glaucoma is below that accompanying other conditions, healthy or morbid.

But this conclusion appeared to be open to the objection that the measurements of a cataractous lens furnish no criterion of its size when still clear, *i.e.*, at the commencement, in the great majority of cases, of the glaucomatous process.

To combat this difficulty, I have recorded of late the measurements of those cases only in which the lens remained clear up to the date of excision. I present all the measurements arranged under four heads, together with brief notes of each case.

To avoid all fallacy due to *post-mortem* change, I have, in almost every case, made them immediately after enucleation. But the measurements made after treatment with Müller's fluid for short periods coincide well with the others, and appear to show that this reagent produces no great, or at least no rapid, alteration in the size of the lens.

In drawing conclusions from these measurements, I have assumed that a lens transparent at the time of excision has at no previous period exceeded its then measurements.

TABLE I.—*Clear Lenses in Eyes the Tension of which is Normal or Subnormal.*

(Twelve Cases, arranged in two groups, of which the first, including five Cases, approaches more nearly the normal condition than the second.)

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 548. Mary G. R. eye excised on account of epithelioma corneæ. T.n. .. .. .. | 62 | 9·75 × 3·8 |
| No. 753. James H. L. eye excised on account of sarcoma, which occupied its outer half from near the optic disc, up to and including the ciliary body. T.n. .. .. .. .. | 58 | 9·65 × 5 |
| No 580. James L. L. eye excised on account of sarcoma of the choroid. T. slightly subnormal, but not − T. 1. (The lens remained 4 days in Müller's fluid before being measured.) .. .. | 52 | 9·1 × 3·75 |
| No. 553. Thomas T. R. eye projected forward by a cyst in the orbit. T.n. No p.l. .. .. | 22 | 9·0 not recorded |
| No. 577. Edward L. R. eye excised on account of a sarcoma behind it. V = $\frac{20}{20}$. T.n. .. | 18 | 8·9 × 4·1 |
| No. 754. Charles C. L. eye has detachment of retina at the macula. New growth (? myxoma) in optic nerve. The ant. chamber is deeper than normal, and the refraction is highly myopic. The iris and ciliary body give pathological evidence of previous glaucoma, though the tension was normal at excision. .. .. .. | 50 | 9·9 × 4 |
| No. 812. John F. R. eye has an infiltrated corneal ulcer from a blow with a twig 2 weeks before excision. Ant. chamber full of pus. Optic disc hazy. .. .. .. .. .. | 57 | 9·8 × 5 |
| No 671. Thomas B. L. eye excised on account of extensive ulceration of cornea. T.n. .. .. | 28 | 9·4 × 4·5 |
| No. 770. Harriet S. L. eye has ulceration of cornea, with severe pain. T.n. .. .. .. | 44 | 9·4 × 4 |
| No. 809. William E. R. eye was inflamed 3 years ago, and has been defective ever since. 8 weeks ago a finger was thrust against it. There is a | | |

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| corneal leucoma with synechia posterior. Tension very slightly subnormal. .. .. .. | 60 | 9·15 × 4·5 |
| No. 682. James P. R. eye had + T. with ulcus corneæ and hypopyon 4 months ago. At excision the cornea was leucomatous, and the T. was normal. .. .. .. .. .. .. | 33 | 9·1 × 4·75 |
| No. 570. Samuel C. L. eye excised, on account of tumour of optic nerve. T.n. at excision. The clear lens is soft and yellowish in tinge. No. p.l. .. .. .. .. .. .. | 65 | 8·8 × 4 |

TABLE II.—*Clear Lenses in Eyes the Tension of which was increased at Excision.*

(Fifteen Cases.)

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 759. Harriet G. R. eye excised on account of glaucoma recurring some time after the tension had been relieved by an iridectomy 2 years ago. The globe is rather large. The ant. chamber is of good depth, and the optic disc deeply cupped. The lens is opaque at the posterior pole only. It has several points of adhesion to the pupillary margin. .. .. .. | 56 | 9·8 × 5 |
| No. 734. William S. R. eye excised on account of a painful glaucoma occurring one year ago. T. 2 at excision. It was iridectomized with good result seven years ago. The lens is yellowish, and has a slight opacity near its posterior pole. .. .. .. .. .. .. | 73 | 9·5 × 4·5 |
| No. 696. Mary P. L. eye excised on account of sarcoma springing from choroid at outer equator, and just encroaching on ciliary body. T. 1. (The lens was in Müller's fluid two days before being measured). .. .. .. .. .. | 49 | 9·5 × 3·7 |

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 787. Joseph P. L. eye of normal size, except that the cornea is staphylomatous at its lower part, the bulging being lined by the stretched and thinned iris. It first failed spontaneously 20 weeks ago. T. 3. .. .. .. | 62 | 9·5 × 3·7 |
| No. 618. William O. Glaucoma of several years' duration. T. 3. .. .. .. .. .. | 54 | 9·5 not recorded |
| No. 850. Ann W. Iridectomy in both eyes for glaucoma 13 years ago, with good result. No trouble till R. became again glaucomatous five weeks ago. The lens is pressed against the cornea. The ciliary body is much atrophied, so as to leave a wide circumlental space. The iris periphery is adherent to the cornea. The vitreous is detached from the posterior pole and shrunk to about one-third its normal size. The retina is detached in loose folds. A sarcoma the size of a large pea extends from near the disk to the equator. It is many mm. removed from the lens. .. .. .. .. | 43 | 9·4 × 3 |
| No. 788. Sarah B. R. eye excised for painful ulcer of cornea, with hypopyon of 5½ weeks' duration. T. 2... .. .. .. .. | 68 | 9·4 not recorded |
| No. 538. John M. L. eye injured 30 years ago by a powder explosion. It remained defective till two weeks ago, when it became watery, red, and painful. At excision T. 2. Retina detached and partly contracted. Ant. chamber is 2 mm. deep.. .. .. .. .. .. .. | 37 | 9·3 × 4·75 |
| No. 675. Eliza G. R. eye excised for glaucoma of one year's duration. Iridectomy upwards was done one month ago, and sclerotomy two weeks later. The iris prolapsed into the sclerotomy wound, which became staphylomatous. An iridectomy downwards was then made without effect. T. 2 at excision. .. .. .. | 46 | 9·3 × 4·5 |
| No. 571. John F. R. eye excised for glaucoma, apparently of but five months' duration. The eye was wounded five months ago, and the cornea is now leucomatous. Measurements of globe: ant. post. 24·5 mm.; horizontal 28; vertical 23·5; O.D. hazy and somewhat swollen; veins tortuous; lens quite clear and *in situ*. The horizontal (9·5 mm.) exceeded the vertical diameter of the lens (9·1 mm.). The mean is | | |

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| given in the table. The lens was found to be more spherical after several weeks' immersion in Müller's fluid, each diameter being 9·05, and its thickness 4·2 mm. The astigmatism that must have resulted from the unequal tension of the lens is of interest in this case. .. .. | 36 | 9·3 × 4 |
| No. 672. Mary B. L. eye glaucomatous three years, with severe pain. T. 2. Cornea steamy; pupil dilated; optic disc cupped.. .. .. | 41 | 9 × 5 |
| No. 842. James P. R. eye faded gradually five years ago from glaucoma. Suppurative iritis came on spontaneously one week before excision. The globe is of normal size. .. .. .. | 67 | 9 × 4·6 |
| No. 685. Clara S. L. eye glaucomatous one year. T. 3. Lens clear, except for posterior polar opacity. .. .. .. .. .. | 47 | 8·75 not recorded |
| No. 563. William C. R. eye bad three weeks with gumma of iris, in consequence of primary sore 5 months ago. T. 1 .. .. .. .. | 57 | 8·5 × 4 |
| o. 663. Robert S. R. eye injury one month ago, followed by hypopyon ulcer of cornea. Saemisch incision, followed by prolapse of iris. .. .. | 46 | 8·5 × 3·5 |

TABLE III.—*Cataractous Lenses in Glaucomatous Eyes.*

(Seven Cases.)

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 537. James B. R. eye dim for 18 months, inflamed and very painful for seven months. Six months before excision there were numerous corneal infiltrations with hypopyon and T. 1, for which an iridectomy upwards was made. A Saemisch incision was made one month later. At excision the lens was close against the cornea; its hard nucleus, measuring 9 × 4 mm., was surrounded by much soft matter. .. .. | 59 | 11 × 5·5 |

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 441. George S. R. eye has been fading for two years. Two months ago it became extremely painful. T. 1. Ant. chamber shallow. Lens opaque; it looks white and glistening, but is quite soft within its capsule, which is evidently distended. It looks thick in its antero-posterior direction. Measurements of globe 23 × 23 × 23. .. .. .. .. .. .. | 73 | 10·5 not recorded |
| No. 640. Emma S. L. eye has been painful occasionally for five years, but much more so for the last three months. T. 2. Ant. chamber very shallow. The lens is of a peculiar translucent whiteness, and looks large and globular. Its capsule is clearly distended, and its contents are semi-opaque and soft.. .. .. .. | 40 | 9 × 7 |
| No. 568. William B. R. eye accidentally found to be blind six months ago. T. 1. The diagnosis is chronic glaucoma. The specimen was 20 hours in Müller's fluid before the measurements were taken .. .. .. .. .. | 57 | 9 × 4·75 |
| No. 610. John L. R. eye blind six years from glaucoma. T. 2. There is a large staphyloma at the equator. .. .. .. .. .. | 45 | 9 × 4 |
| No. 560. John P. L. eye glaucomatous six years. T. 2. Ant. chamber 4 mm. deep. Iris tremulous; its periphery adheres to the cornea. The lens margin is removed 1 mm. at least from the apices of the ciliary folds, which, with the ciliary body, are shrunken. .. .. .. | 44 | 8·5 × 3·5 |
| No. 602. Richard B. R. eye has been blind many years, and painful occasionally for four years. The lens is distant 1·5 mm. from the cornea. It is about 2 mm. removed from the atrophied ciliary folds and ciliary body. The retina is detached. T. 3. .. .. .. .. .. | 50 | 8 × 4·75 |

## Table IV.

(Five exceptional Cases, with increased tension.)

| | Age of patient. | Measurements of lens. |
|---|---|---|
| | | Diameters. lat. ant. post. |
| No. 761. Edward D. Eight years ago L. eye became totally blind in the course of 12 months. At excision T. 2. The lens, opaque at its centre and poles, is partially dislocated upwards behind the iris. .. .. .. .. .. .. | 64 | 8·9 × 5·5 |
| No. 531. Thomas I. L. eye has kerato-iritis of very long duration with excluded pupil. Lens semi-opaque. .. .. .. .. .. | 19 | 8·75 × 2·75 |
| No. 549. George N. L. eye blind from a blow many years ago. T. 1. Total posterior synechia. .. .. .. .. .. .. | 40 | 8·75 × 4·5 |
| No. 635. Mary B. R. eye blind from a blow with a piece of wood three months ago. Lens partially dislocated downwards behind the iris. T. 3. .. .. .. .. .. .. | 63 | 8 × 5·5 |
| No. 539. Thomas C. Glioma retinæ of R. eye. Lens clear. T. 1. .. .. .. .. .. | 4 | 8 × 4·25 |

The results are as follows :—

| | Age. | Diameter. |
|---|---|---|
| Averages deduced from Table I :— (Transparent lenses. Normal or subnormal tension.) | | |
| First group .. .. .. | 42·4 | 9·28 |
| Second group .. .. .. | 48·1 | 9·36 |
| Or both groups taken together .. | 45·75 | 9·33 |
| Averages from Table II .. .. .. .. .. (Transparent lenses of glaucomatous eyes.) | 52·1 | 9·21 |
| Averages from Table III .. .. .. .. .. (Opaque lenses of glaucomatous eyes.) | 52·6 | 9·3 |
| Averages from Table IV .. .. .. .. .. (The cases of increased tension not included for various reasons in Tables II and III.) | 38·0 | 8·5 |

| | Age. | Diameter. |
|---|---|---|
| But if we take the cases where the age exceeds 50 years separately, we get— | | |
| (*a.*) Averages deduced from three cases (Nos. 548, 753, and 580) of Table I. T. n. .. .. .. | 57·0 | 9·5 |
| (*b.*) Averages from seven cases of Table II. T. in excess .. .. .. .. .. .. | 62·4 | 9·3 |
| If we include also with these last the cases from Table III which belong to the same period of life, we get— | | |
| (*c.*) Average from ten cases of glaucoma .. .. | 62·6 | 9·57 |

It will be noticed that even this last-mentioned diameter is not very different from 9·5, the apparent normal diameter at a rather younger period of life, notwithstanding that the two most striking of the exceptional cases (Nos. 537 and 441) are included. It is to be admitted, however, that the presumably normal lenses are but few in number.

It is clear that if the cases of Table IV had not been excluded from Tables II and III, the resulting average diameter of the lens in glaucoma would be very considerably, and probably even unduly, below the normal. The causes of their exclusion will be found in the brief notes attached to each case.

From these results of the above measurements, each made with great care and under the observation of several persons, we shall probably be justified in concluding that in glaucoma an enlargement of the lens is an uncommon and an unimportant occurrence, and that its average diameter in this disease, whether it be cataractous or clear, is even somewhat below the normal at the corresponding period of life.

With regard to the three remarkable cases of glaucoma, in which the lenses were very large (Table III, Nos. 537, 441, and 640), two only of them come within the period during which the remaining measurements were collected. One of these, however (No. 537), is not entirely free from the suspicion that the enlargement may be due to a traumatic influence.

I am disposed to attribute these comparatively rare cases to a stage in the development of cataract or perhaps rather of a certain form of cataract, occurring in glaucomatous eyes, as

in others. For the lens matter is not only swollen, but also soft and semi-opaque. A hard nucleus is present in only one of the three cases.

As far as I know, there is no pathological evidence that such a stage exists. It has long been suspected from the condition of the anterior chamber and from changes in the refraction. Becker* regards its existence as conclusively established upon clinical observation alone. Certainly the anterior chamber was encroached upon by the enlarged lens in the cases I have recorded here.

Clinical observations frequently show an advance of the lens in glaucomatous eyes, and this advance may be demonstrated *post-mortem* by properly prepared specimens in suitable cases. But in other cases of glaucoma, usually of longer standing, the lens, though probably advanced earlier, is found occupying its normal position. The anterior chamber, however, usually retains its shallow appearance from the peripheral union of iris and cornea.

The vitreous body is clearly the immediate agent of the advance of the lens, for detachments of this body by means of accumulations of fluid in front of the ora serrata, appear to be as rare in glaucoma as in other diseases.

When we find the vitreous fully occupying its chamber, we must attribute the advance of the lens to its increased size, just as the pushing back of the lamina cribrosa is due to the same cause. When, on the other hand, the fluid accumulates behind the vitreous in the subhyaloid space, we must attribute the position of the lens to an insufficient passage of fluid into and thence forwards through the shrunken vitreous. If the fluid is in the subretinal space, the retina itself must be the seat of delay.

The accumulation of fluid either within or behind the vitreous must be due either to the separation of an abnormally large quantity from the blood-vessels surrounding it, without a proportionate increase of that escaping from it into

* Handbook of Graefe and Saemisch, vol. v.

the aqueous chamber, or to an insufficient drainage in the last-mentioned direction, the quantity passing from the blood-vessels remaining unaltered.

The theory that a mechanical obstruction at the anterior surface of the vitreous prevents the outflow there of the usual fluids, and thus causes the advance of the lens, ciliary folds and iris, has been a favourite one, and the obstruction has naturally been sought at the region of greatest physiological flow, *i.e.*, close external to the lens margin.

If a mechanical obstruction checked the outflow here, we should expect to find an accumulation of fluid immediately behind it, that is to say, between it and the vitreous, just as we find the posterior chamber distended when the pupil is excluded.

The subject of anterior detachments of the vitreous has not received sufficient attention. Pagenstecher* records eight cases of enlargement of the canal of Petit, and consequent detachment of the vitreous in the ciliary region. One case of ciliary detachment is figured by Auquier.† Several have also come under my own observation. But none of them furnish any evidence that this condition bears any special relation to primary glaucoma.

It might, however, be said that fluid, checked by obstruction at the anterior surface of the vitreous, would, on account of the less firm anterior adhesions of that body, less easily accumulate here than in the vitreous itself. But even this could not account for the rarity of accumulations in the canal of Petit.

We may, however, leave this part of the question, and ask—What proof have we that such a mechanical obstruction ever exists ?

I have set myself to show earlier in this paper that physiological variations in the size of the lens are not sufficient to produce one. And if, as would appear to be the case, certain forms of cataract not commonly or necessarily associated with

* A. f. O., xxii, 2.

† Du décollement hyaloïdien. Paris, 1878.

glaucoma (*see* page 379), begin with a considerable swelling of the lens, we have an argument of considerable force.

The occasional cases in which increased tension rapidly follows traumatic swelling of the lens require further investigation. Such cases, especially if the lens be pressed against the cornea, are the only ones in which it would appear even probable that the tension is due to mechanical obstruction by the enlarged lens. We must remember on the other hand, that such swelling can hardly constitute the entire morbid change in the eye, and that comparatively few such injuries are followed by tension.

Moreover, the lens would require to be very considerably increased in diameter before it could by itself constitute an obstruction. For the surplus fluids of the vitreous chamber pass forwards, not between the lens and the summits of the ciliary folds, for the two touch under normal circumstances, but along the channels between the folds. And as these channels are ·75 mm. in depth, measuring from the lens margin to the ciliary body itself, and only ·25 mm. in width at their widest, that is, their most peripheral part, it is clear that any influence exercised by the lens would be insignificant in comparison with that produced by the swelling of the ciliary folds themselves.

But we lack pathological evidence of any enlargement of these latter. On the contrary, my observations distinctly lead to the conclusion that their average size is greater in eyes with normal or sub-normal tension than in glaucomatous eyes. Indeed, it seems indisputable that their size is directly influenced by the intra-ocular pressure, and it is highly probable that one of their functions, especially of their anterior or most projecting part, is to adapt themselves to, and to some extent regulate, the varying conditions of intra-ocular pressure.

But if there is no mechanical obstruction at the anterior surface of the vitreous in glaucoma, how do we account for the subsequent recession of the lens and for the more common occurrence of this disease, especially when associated

with shallowness of the anterior chamber, at advanced periods of life, when, according to the interesting researches of Priestley Smith,* the lens would appear to attain its maximum size?

The latter part of the question, which would appear at first sight to be associated with the size of the lens, might be explained by the greater elasticity in children of the tunics of the eye, which permits them to yield temporarily or permanently to their increased contents. In this way the increasing contents of the vitreous chamber do not require to press so much upon the lens, which in its turn has greater power of resistance owing to the greater tension of the zonula. Thus the dangers attendant on the contact of iris and cornea are frequently averted. Such cases often present themselves, in my opinion, as cases of progressive myopia.

And the recession of the lens at the later periods of glaucoma, which forcibly calls to our minds the atrophy of the ciliary body and consequent widening of the circumlental space, would be perfectly explained by the more effective obstruction which must, for the glaucoma to become permanent, have become established at the periphery of the anterior chamber, one which no increase of pressure can overcome, whereas the outflow at the zonula appears, according to Weber's recent most admirable exposition of the subject,* to be increased by the augmenting pressure behind it over that in front; the general result of the complete block at Schlemm's canal, the partial one behind, and the diminished inflow due to the ciliary atrophy and the pressure on the uveal blood-vessels, being that the pressure in the aqueous and vitreous chambers becomes equalized, and the lens regains its position of equilibrium. It may also be noted that a glaucoma cured by iridectomy may subsequently recur with a second advance of the lens.

Nor is there any proof that the lens offers any undue resistance in glaucoma to the passage of fluid through it. For cataract extraction is sometimes followed by increased

* Trans. Internat. Med. Congress. London, 1881.

tension with the characteristic advance of the iris and apposition of its peripheral part to the cornea.

Moreover, extraction of the lens from a glaucomatous eye does not seem to be more efficacious in preventing the recurrence of the tension than a simple iridectomy. And backward dislocations of the lens, whether complete or partial, are often followed, and sometimes with great rapidity, by glaucoma.

On these various grounds I fail to recognise in cases of primary glaucoma any obstruction to the normal outflow of fluid at the anterior surface of the vitreous sufficient to advance the lens. If we must have obstruction as a cause we must look for it in the vitreous itself. But we find no evidence of this, at least in the cases where this body is not shrunken or altered in structure. The same may be said with regard to the retina in relation to the accumulations found behind it, not only with choroidal tumours, but in many advanced cases of primary glaucoma.

If the increased contents of the vitreous chamber cannot be attributed to a diminution of its normal outflow, we must fall back upon the theory first advocated by Donders, of an increased separation of fluid from the blood, *i.e.*, an exaggeration of the normal process.

It would be proceeding beyond the purpose of this paper to discuss the evidence in favour of this view. It must always be difficult to produce pathological proof of a functional disturbance.

Suffice it to say, that it rests rather on the absence of contrary evidence than on positive proofs. Certainly in primary glaucoma the pigment cell layer of the ciliary body, which is presumably the principal agent recruiting the vitreous and aqueous chambers, is not the subject of any structural change which would appear to destroy its power in this direction, whereas such changes are common in the secondary glaucomas which are clearly due to obstruction at the corneal margin, *e.g.*, after perforating ulcers.

And I have found, even in the course of my usual micro-

scopic examinations, the pars ciliaris retinæ detached from the subjacent pigment layer in about 10 per cent. of the cases of primary glaucoma. These detachments are frequently so localized as to give the appearance of little fluid-containing cysts, but sometimes they are of considerable extent. Very similar changes are found in association with the cyclitis found accompanying the disease known clinically as Iritis Serosa, and which therefore should, I presume, be called Cyclitis Serosa. In this condition, however, the cysts contain some corpuscular elements. I have never found this cystic appearance of the pars ciliaris retinæ associated with normal or subnormal tension.

Does it indicate that fluid is secreted by the pigment cell layer more rapidly than it can pass onwards into the vitreous chamber ?

It is very commonly associated with so-called cystic disease of the anterior part of the retina, a condition which, though it may present itself as a senile change, is more commonly and more strikingly associated with glaucoma. Further investigation, however, is required in order to determine the precise significance of these changes and also of the enlarged afferent vessels of the ciliary body.

## HOUSE SURGEON'S NOTES.

By W. J. Milles, F.R.C.S.,
*Senior House Surgeon.*

### *Iodoform in Purulent Ophthalmia.*

I have employed this drug for some time past in purulent affections of the conjunctiva and cornea, and I believe that, in suitable cases, it is of very great service.

The best method of using it is either in a very fine powder, when care should be taken that it is ground down very thoroughly; or in the form of ointment of variable strength, the usual form being gr. xxx to ℥j of vaseline, the stronger form to ʒj to ℥j. The importance of iodoform in ophthalmic practice is that it produces less pain and irritation than any other antiseptic that I am acquainted with; that it can be used in a concentrated form; and that it volatilises very slowly.

Eighteen months ago I gave it an extended trial in ophthalmia neonatorum, and came to the opinion that it was only useful when one could treat the disease from the commencement (within the first two or three days). In the later stages when there was thickening of the lids, and a granular condition of the conjunctiva, I found that its effects in stopping the purulent discharge were very slight. I was more pleased with iodoform in the treatment of gonorrhœal ophthalmia, possibly from the fact that the patients suffering from this affection were more immediately under control. The treatment was combined with the use of boracic acid lotion, or a solution of perchloride of mercury (gr. i to ℥j), and, of course, a constant application of iced compresses. In a number of cases the acuteness of the swelling and the profuse discharge rapidly diminished, and the cornea was saved under this form of treatment.

Having seen the rapidity with which an indolent ulcer of the leg changes its character under the use of iodoform, I was led to try its effect on the slowly-spreading infiltrated ulcer of the cornea. It quite answered my expectations. In several cases (though other treatment had been tried without effect) with the constant application of iodoform, the ulcers ceased to spread, the infiltration of their edges rapidly disappeared, and a rapid recovery ensued.

### *Atropine Ointment.*

The ordinary atropine solution, as I have frequently found, becomes turbid in a few days from the growth of micro-organisms. It is naturally an objectionable form of treatment to bring such a solution into contact with a recent corneal wound, whether produced surgically or otherwise. For this reason I have ceased using atropine in this form for the treatment of in-patients; and I believe that an ointment made by combining gr. iv—viii of sulphate of atropia to ʒi of vaseline is a much more satisfactory method of application.

Besides being practically aseptic, it has other advantages. It can be applied to the outer surfaces and edges of the closed lids with perfect efficacy. This fact I have tested frequently and consider to be of some importance, especially in the after-treatment of cataract operations, where it is of great importance to disturb the eyes as little as possible. Again its action is more constant than that of the solution, and lasts for a longer period. This point was shown very clearly in a case of interstitial keratitis with much photophobia and lacrimation, for directly the atropine solution was dropped into the eyes, a flood of tears washed it away, and its action was *nil;* one application of the atropine ointment dilated the pupils fully.

### *Spasmodic Entropion of Lower Lid.*

This very troublesome affection is not an uncommon occurrence after operations on the eyes, and keeps up a great

amount of irritation. I have found that the ordinary method of treatment, *i.e.*, traction downwards of the lower lid by adhesive plaster is, as a rule, next to useless, for the lacrimation produced by the entropion soon destroys its action. The removal of a piece of skin from the lower lid of course relieves the patient in a day or two; but if the trouble, which is only temporary, can be relieved without an operation, so much the better.

The following plan proves successful. Take a piece of fairly thick soft lead wire, as is often used for styles. Mould it as accurately as possible to the face by commencing from the centre of the forehead, bringing it halfway down the middle of the nose, thence at right angles along its side, and just below the lower lid to the prominence of the malar bone; again at right angles, and now parallel to the first part to the front of the temple. Strap the two ends of the wire firmly to the forehead, and apply a bandage over the strapping. A little manipulation of the wire to the extent of lightly indenting the skin just within the lower orbital ridge, will keep the lid well everted.

A piece of thin sheet lead, shaped something like the ordinary strabismometer, often acts equally well.

### *Iridotomy.*

A simple modification of this operation suggested itself to me, and seems to possess some advantages.

It consists in making the corneal incision in a radial manner, the outer end being at the corneo-scleral margin; that is to say, it is at right angles to the ordinary incision in the circumference of the cornea. The resulting scar is of course far external to the axis of vision.

The advantages of this modification that I have noticed are—

1st. That the incision in the cornea can be much more limited, and proportionally gives more play to the scissors.

2nd. That the knife can be plunged directly through the iris in the direction previously decided.

3rd. That the scissors are introduced and remain in the same direction, and, consequently, that they completely fill up the corneal incision and so prevent any escape of vitreous.

In the ordinary operation the scissors have to be turned through a quarter of a circle, and necessarily make the incision gape to its utmost extent, thus allowing a free escape of vitreous.

## CURATOR'S PATHOLOGICAL REPORT.

By W. A. Brailey, M.D.

*Cases Illustrative of New Formations in or upon the Choroid.*

No. 730. Augustus D., æt. 39. Excision of *right* eye. The microscopic specimens were prepared by Dr. Lobo.

The *left* had experienced sympathetic irritation during the last four weeks.

The *right* was wounded with glass 30 years ago. Since then, though sightless, it has been free from pain.

*At excision*, umbrella-shaped detachment of the retina. In or on the internal surface of the choroid there is a bony layer, studded at intervals on its retinal aspect by small nodules, as if of colloid disease. Most of these are due to rounded elevations of the subjacent bone, with its covering fibrous layer. But others are made principally of yellowish granular matter, apparently altered blood, surrounded by a slightly fibrillated, but otherwise structureless outer wall. They are rounded in form, and generally pedunculated. The pigment epithelial layer is degenerated or wanting.

The bone is disposed in an irregular reticulum, formed by bands disposed at different levels, and uniting at various points. The interspaces between them contain a very delicate connective tissue, and each has at its centre a large, extremely thin-walled vessel (diam. ·04—·12 mm.), evidently an enlarged capillary.

As a rule the vessels of this layer of new formation are closely packed with blood corpuscles, among which the colourless form is unduly abundant. The blood-vessels of the subjacent choroid present this change less frequently, and in a less marked manner, though the walls of the arteries are often much increased in density and thickness.

There are, however, some areas of the choroid over which bone is absent, and the newly-formed layer in or on its internal surface consists entirely of fibrous tissue, whose dense fibres are arranged in parallel planes. But it may be that some of these fibres are replaced by bands of very much denser tissue, evidently

a stage of development into bone; while we have also occasional tracts of looser tissue, probably corresponding to the interspaces which will, when the development is completed, contain delicate connective tissue and blood-vessels. These changes into bone take place especially in the external (deeper) layers of the newly-formed fibrous tissue, so that a layer of vascular sparsely nucleated fibrous tissue of varying thickness comes to lie on the internal surface of the bone.

The connective tissue, which, with its contained capillary vessel, may fill up the interspaces of the bony reticulum, may be replaced by a yellowish granular globe (diam. ·12 mm. or more) within a hyaline or fibrous ring, the whole being very much like some of the nodules already described as projecting into the sub-retinal cavity. They appear to be capillaries which have become plugged and very much distended. Next externally to the bone-containing layers, and extending from them up to the sclerotic, is a stratum which evidently represents the choroid, for it contains a few capillaries, some larger vessels, and branching pigmented cells. Moreover, it is separated from the bone-containing stratum by a thin undulated membrane of uniform thickness, evidently the lamina vitrea of the choroid.

Thus the bone, which is recognised by its hardness, and by the presence in its structureless matrix, after maceration in chromic acid solution, of many angular brightly refracting corpuscles, is entirely on the inner choroidal surface. It is probably inflammatory in its origin, though evidence of this is generally wanting. At two spots, however, the chorio-capillaris is densely packed with inflammatory cells.

There is a fibrous layer on the posterior surface of the iris, and one also on the posterior surface of the detached retina, near the ora serrata. The latter layer contains a small amount of bone.

No. 762. William B., æt. 32, under care of Mr. Lawson. Excision of left eye. Microscopical specimens prepared by Dr. Charnley.

*Right* eye suffered from sympathetic irritation of two months' duration.

The *left* received a blow nine years ago. The sight went entirely away in the course of 1½ years. It has been painful for the last two months.

Globe rather shrunken. Pupil small, occluded. Iris discoloured. Ant. ch. very shallow. The retina is detached from the choroid, except just round the optic disc, where a circle of bone lies between and unites the two. The sub-retinal fluid contains crystals of cholesterine. There is also an irregular circle of bone extending all round the posterior termination of the ciliary circle.

Microscopic sections through one part of the bony ring, round the papilla, show that it rises rather steeply from the choroid into a hillock, ·75 mm. high, its diameter at the base being 1·3 mm. It is covered on its internal (retinal) surface by a vascular fibrous layer, rather thick at the summit, whence it shades off gradually towards the sides, till it soon disappears on the adjacent unaffected choroid. Underneath this fibrous layer the several imperfect layers of bone, of which the hillock is mainly made up, unite with one another irregularly. The interspaces between them contain thin-walled blood-vessels, probably dilated capillaries, embedded in very delicate connective tissue. The whole is separated from the subjacent choroid by a thin, homogeneous, much undulated membrane, probably the vitreous lamina. Beneath it are a few capillaries, probably those of the chorio-capillaris, and, deeper still, the larger choroidal vessels. The tissue of the choroid at the seat of the new formation is a little thinner than elsewhere.

The other bony ring is situated on the internal surface of the choroid, immediately behind the ora serrata. It lies in the midst of a fibrous layer which, gradually diminishing in thickness, runs for some distance along the internal surface of the adjacent choroid. In places a very distinct undulating thin lamina can be seen to separate the fibrous and bony layers from the choroid proper. This separation becomes much more distinct as we pass backwards.

No. 782. Wm. R., æt. 34, under care of Mr. Couper. Excision of left eye, on account of recent sympathetic irritation. Microscopic specimens prepared by Dr. Lobo.

The eye was wounded thirty years ago, and has had no perception of light since. It is shrunken to two-thirds its size. Its cavity is nearly filled by a bone-containing mass, situated in or on the choroid. A tough, fibrous, non-vascular layer bounds this mass internally, and separates it from the contracted retina.

The total thickness of the new formation is about 3 mm. Its relations are the same as in the specimen No. 730. Its structure is also the same, except that its connective tissue shows a few stellate pigment cells, and that its inner fibrous stratum is not quite so inconsiderable relatively to the entire thickness of the new formation.

In the deepest part of the mass, or, more often, on its external surface (*i.e.*, that near the choroid proper), we encounter many large bodies (diameter about ·01 mm.) which in section show an outer, more or less thick, structureless, opaque wall, enclosing a central cavity, which is plugged with yellowish granular matter. These appear to be dilated and obstructed blood-vessels, and they remind us of the bodies met with in Case No. 730, in the choroid, and also in or on the newly-formed layers internal to it. Their size and general disposition are much like those of the immediately adjacent bony rods, and it does not appear altogether improbable that the latter were originally derived from the conversion of blood-vessels and their contents into bone. The anastomosing rods of bone are loosely arranged, and their larger interstitial spaces are occupied by extremely delicate connective tissue, in which are found small arteries with much-nucleated walls, and also a moderate number of very fine capillaries. The stratum, which appears to represent the choroid proper, though denser and containing more pigment, is on the average only ·2 mm. thick, and contains very few blood-vessels of any description. It is separated in places from the bone-containing layer by a delicate-looking undulated lamella, which appears to represent the remains of the internal limiting membrane of the choroid (lamina vitrea).

The ciliary body is clothed on its inner surface by a tough membrane, which is pigmented in its deeper part. A similarly pigmented fibrous layer, containing some bone and a few blood-vessels, lies on the anterior part of the choroid, and is bounded on its external surface by a thin, structureless-looking, much-undulated lamella, which can be traced as a direct continuation of the outermost of the two lamellæ which normally separate the connective tissue of the ciliary body from the overlying pigment cell layer and pars ciliaris retinæ. This continuity of the membrane, coupled with the presence of pigment in the anterior part of the newly-formed stratum, makes us yield to the

belief that the latter, both here and further back, is situated on the *internal* surface of the well-defined lamella, which limits the choroid internally (the lamina vitrea, as commonly seen). The anterior part of the choroid is thick and fibrous in its external layers.

No. 736. John P., æt. 34, under care of Mr. Tay. Excision of *right* eye, which is shrunken from corneal necrosis after gonorrhœal ophthalmia, beginning four months ago.

The cornea can scarcely be traced; the retina is detached; the choroid is *in situ*. In two places near the equator we encounter, near the junction of sclera and choroid, a well-defined rounded cavity, which contains dark blood. Such cavities have quite a cyst-like appearance, but their continuity can be demonstrated by the aid of a probe. Their course is very extensive, and they give off ramifications.

Microscopic sections, prepared by Dr. Lobo, show that the main cavities have smooth walls, and are situated in the midst and parallel to the surfaces of a thick fibrous stratum, which occupies the position of the lamina supra-choroidea. This stratum is separated from the sclerotic by a loose layer of branching pigment cells, which is sometimes split by means of an intervening cavity into two secondary layers, which evidently represent the pigment of the lam. supra-choroidea and lam. fusca. The cavity consequently represents the peri-choroidal lymph space. It may be of very considerable size, but this is probably due mainly to the process of preparation, and it is doubtful if it ever produces the cyst-like appearance described as visible in this specimen to the naked eye.

The fibrous stratum (the supra-choroid) is as much as ·5 mm. thick, whereas the remainder of the choroid to the very summits of the undulations of its internal surface measures only about ·3 mm.

The sclerotic and supra-choroid, though both fibrous, contrast strongly in appearance, for the fibres of the latter are parallel to its surfaces, and show numerous nuclei, whereas those of the sclerotic, as is commonly the case in atrophied eyes, are sparsely nucleated, and very much undulated.

The remainder of the choroid is diffusely hypernucleated, and contains an excess of dark brown branching pigment cells.

The lamina vitrea is much undulated, but not at all thickened. The pigment epithelium of its internal surface is very irregular, but its pigment is generally unduly abundant and dark in colour. There is no new formation on the inner choroidal surface.

No. 737. Fredk. D., æt. 32. Excision of *right* eye. The *left* looks perfectly healthy, but has watered a little at times for many years past.

The *right* was lost more than ten years ago, from a spontaneous inflammation, apparently an iritis. It has been painful occasionally for several years, but much more so within the last fourteen days. It is somewhat shrunken. There is ciliary injection. The atrophied iris is thrust against the cornea. Perception of light is wanting. After excision we find umbrella-shaped detachment of retina and sub-retinal space entirely filled by a mass which has the same structure and arrangement as in Specimen No. 782.

In many places we can trace on its external surface a thin, uniform, much-undulated membrane, evidently representing the choroidal vitreous lamina. The choroid proper, which underlies this, is, in its external layers, very dense and fibrous, so that the lamina supra-choroidea looks not unlike the adjacent sclerotic. But the two are separated in places by an extensive interspace, probably the peri-choroidal space. The rest of the choroid is thinner than usual, and contains more fibrous tissue. Its vessels, comparatively few in number, have thick walls. The microscopical specimens are not very good.

No. 217. John J., æt. 24. *Right* eye excised by Mr. Lawson. The *left*, which was always very myopic and otherwise defective, was injured ten months ago by a small piece of steel. It has been painful for the last two days.

The *right* has always been extremely defective. He knows of no cause for this. It never squinted. An iridectomy was made in it nine months ago. *At excision* its tension is diminished: there is a large artificial coloboma outwards. The retina is detached: there is a thin pale yellow fluid between it and the choroid, which remains *in situ*. The optic disc is surrounded by a bony plate through which the pedicle of the

detached retina runs. There are other smaller patches of bone on the inner choroidal surface, as well as a very imperfect bony circle just behind and parallel with the ora serrata.

Meridional sections of the ciliary body and iris show a considerable increase of inflammatory cells in their connective tissue, especially near the junction of the two. These are also very abundant round Schlemm's canal. In the anterior part of the choroid and adjacent posterior part of the ciliary body they are arranged in masses something like those of uveitis maligna. On the internal aspect of the posterior part of the ciliary body and adjacent choroid is found a fibrous layer, which, *in front of the ora serrata*, bears the remains of the pars ciliaris retinæ on its internal surface, and in its deeper layers many pigment cells. The same *behind the ora serrata* contains some pieces of bone. A homogeneous structureless lamina, evidently the lamina vitrea, separates the bony and fibrous mass from the choroid proper. The presence of the newly-formed fibrous and pigmented layer distinguishes this inflammation from the typical choroiditis maligna of sympathetic disease.

At another point of the ciliary circle the same changes are seen in a less advanced condition. The proliferated pigment cells and many unpigmented ones are disposed in a soft fibrous tissue on the internal surface of the vitreous lamina.

At the *posterior part* of the choroid the bony plates lie in the midst of much nucleated connective tissue. They contain cavities occupied by similar tissue and some blood-vessels. A delicate undulated uniform membrane separates the mass from the subjacent choroid proper, in which many cells are distributed in clusters in a manner which is occasionally slightly suggestive of choroiditis maligna. The sclerotic contains more nuclei than normal.

Judging from the numerous well-developed bone corpuscles, and from the numerous nuclei of the connective tissue in which it lies, it would appear that the new-formed bone is in a high state of nutrition. In this case we see none of the dense fibrous tissue which in other cases lines the bone on its retinal aspect.

No. 821. Wm. B., æt. 39. *Left* eye excised by Mr. Streatfeild, on account of its producing sympathetic irritation. Its sight was lost from a blow five years since, but it gave him no

trouble till seven months ago, when it received a scratch from an iron nail. Since then it has been painful at times.

At excision its size and tension were normal. The shrunken lens was dislocated. The detached retina contained within its cavity a mass of cholesterine, and was separated from the choroid by a fluid containing cholesterine crystals. The choroid is dark in colour, but otherwise appears normal. The upper part of the ciliary body bears on the inner aspect of its posterior half a hard, white, bone-like mass.

Microscopic examination (by Mr. Hartridge) shows that this mass is in reality osseous, containing numerous bone corpuscles. It is traversed by tracts of vascular connective tissue. It lies in the midst of the irregularly proliferated pigment of the ciliary body. The latter is much thinned and atrophied where it underlies the new formation. Elsewhere, though somewhat hypernucleated, it is neither atrophic nor is its pigment proliferated.

No. 566. Wm. T., æt. 14. Excision of *left* eye by Mr. Tay. *Right* normal.

The *left* is shrinking from an injury with a pocket-knife four years ago. Now it is sightless and painless.

The cornea is clear, and the iris discoloured. The lens area is occupied by a white opaque hard plate, resembling bone. The retina is detached, and the sub-retinal space contains a pale yellowish-red fluid in which are cholesterine crystals. The choroid is *in situ;* on its internal surface are several prominent nodules, some even 1·5 mm. high; ridges sometimes connect adjacent ones.

Microscopic sections, by Dr. Lobo, show that the nodules are made of concentrically-disposed, somewhat fibrous, laminæ, between which are numerous bone corpuscles and occasional brown pigment granules. The superficial laminæ form a comparatively unstained zone. Then, as we leave the surface, the staining becomes rather suddenly a little deeper. Towards the centre the fibres are more irregularly disposed and between them, or in a central more or less well-defined cavity, is a yellowish granular matter, intermixed with which are many colourless acicular crystals. These contents are probably the remains of blood.

The nodules, though sometimes sessile, are generally pedunculated, often by an extremely narrow neck. This is sometimes implanted on a bony plate which forms a base, gradually thinning towards its margin, and resting on the subjacent choroid. The latter is generally normal, except that it is a little thinner, and its stroma is rather more pigmented at these spots. Its unchanged lamina vitrea can often be traced as a delicate homogeneous line which separates the choroid from the new formation.

No. 248. Susannah H., æt. 17. *Right* eye excised by Mr. Hulke. The *left* has been irritable for the last six months.

In infancy she had ophthalmia neonatorum which left the *right* blind. She has had no trouble with it till six months ago, when pain came in and over it. At excision it is shrunken and extremely hard (T. 2). The cornea is small and cicatricial, with thinning at its upper margin. The retina is detached and contracted. The internal surface of the choroid is studded with greyish nodules, most of which are sessile by a broad base. Some are as much as ·25 mm. in height.

The microscope shows them to be on the internal (retinal) surface of the lamina vitrea, which is almost everywhere unchanged. Most of the nodules are covered by enlarged and darkly pigmented cells of the pigment epithelium layer. Many stain very deeply and uniformly with hæmatoxylin. But in some this staining is limited to the central part. Others, especially the smaller ones, remain entirely unstained. For the most part no structure can be detected in any of them, though occasionally, large faintly-staining oval nuclei can be seen in them. In two cases, however, the deepest part of the nodule is packed with closely arranged parallel elongated nuclei; and similar nuclei occupy in these cases the corresponding part of the chorio-capillaris, the two being separated by the delicate unaltered lamina vitrea. In one of these cases that part of the new formation which occupies the chorio-capillaris contains some bone corpuscles, so that the colloid nodule is capped on its deep surface by bone situated in the chorio-capillaris.

On the external surface of the pedicle of the detached retina there is a layer of fibrous tissue which contains some small osseous formations. A continuation of the choroidal vitreous

lamina can be traced for some distance on the commencement of this pedicle. In this position it is distinctly thickened, and bears colloid bodies and the remains of pigment cells.

No. 63. Charles B., æt. 23. Excision of *right* eye by Mr. Streatfeild. *Left* sympathetic irritation for the last six months.

The *right* was wounded with scissors sixteen years ago. He has had neither perception of light since, nor any pain.

*At excision* it is rather smaller than the average, though the tension is normal. The retina is detached into an umbrella shape; the choroid, which is *in situ*, has conspicuous nodules projecting from its internal surface. These are found by the microscope to lie between the choroidal vitreous lamina and its pigment epithelium. They are limited to the posterior half of the eyeball, and are comparatively few in number. Their size varies greatly: the largest are ·3 mm. in height, some are pedunculated, but the majority are sessile by a broad base. The cells of the pigment epithelial layer overlying them are often large and unduly dark in colour, but they may be wanting altogether.

The new formation may either show large feebly staining nuclei embedded in a structureless matrix, the whole having somewhat the appearance of a so-called giant-cell; or it may be entirely structureless, in which case there may be a slight concentric lamination. Or its more projecting portion may be structureless, while that part in contact with the vitreous lamina either shows nuclei in a structureless matrix or consists of nucleated fibrous tissue, in the centre of which there is a plate of bone. Thus, we may have a true ossification on the internal surface of the vitreous lamina like in kind, but much less in degree than in the cases previously described.

The new formations take the hæmatoxylin staining very variously, from a weak diffuse coloration to a diffused intensity, which almost prevents the recognition of the enclosed nuclei.

The capacity of staining is retained longer by the central and deepest parts.

The lamina vitrea is not thickened. In the choroid generally there is a moderate degree of hypernucleation, which is rather more conspicuous in the spots whence the projecting

nodules take their rise. Here also the choroid may be slightly thinned.

The generally unatrophied ciliary body presents certain points where its pigment epithelium is thickened and intermixed with newly-formed fibrous tissue. The part of the ciliary body immediately underlying these spots is atrophied so that they form comparatively inconsiderable projections into the vitreous chamber.

The anterior part of the choroid presents many small nodular elevations, to the formation of which the enlarged pigment epithelium cells conduce either partially or entirely.

No. 231. Dinah H., æt. 36. *Right* eye excised by Mr. Hulke, on account of sympathetic irritation. The sight was lost 19 years ago from a blow with a piece of wood, which partially dislocated the lens. Now there is an opacity at its posterior pole, and almost complete absorption of the vitreous. The retina and choroid are *in situ*. They present several pigmented spots, in appearance exactly like those of choroiditis disseminata. Microscopic sections through these spots show that the choroidal vitreous lamina is unchanged, while its pigment epithelium is irregularly thickened and unduly black. In the midst of this hypertrophied epithelium, or on its internal surface, are many large deeply-staining nuclei, apparently those of inflammatory cells of new formation. Sometimes these cells are replaced by sparsely nucleated fibrous tissue, in which case the pigment epithelium cells cannot be traced. The retina and choroid cohere at these points; elsewhere they are separated.

A small piece of bone is found embedded in the choroid in the immediate neighbourhood of the optic disc. The choroidal layers which separate the bone from the sclerotic contain numerous brown pigmented stroma cells; the narrow stratum by which it is separated from the unthickened lamina vitrea contains numerous elongated staining nuclei, arranged parallel to its surface. There is a mass of unpigmented nucleated exudation on the internal surface of the lamina vitrea corresponding to the bone.

No. 189. Solomon N., æt. 42. *Right* eye excised by Mr. Wordsworth on account of sympathetic irritation. The eye is

shrinking after an iritis, which began five years ago. The retina is detached; the choroid remains *in situ*. At one part of the inner surface of the latter membrane is found a dense sparsely nucleated fibrous layer, which is separated from the subjacent partially atrophied choroid by a very delicate, homogeneous-looking, much undulated membrane, which is the lamina vitrea. At other points there are colloid masses, and numerous enlarged, very dark, epithelial cells. The lens is represented by a calcareous-looking shell. A tough pigmented membrane is found at one point only of the internal surface of the ciliary body, representing a very much localised cyclitis. The cause of the inflammation is obscure. It may be syphilitic.

No. 760. John A., æt. 54. Buphthalmic condition of *right* eye since infancy. Tension normal at excision, which was performed by Mr. Streatfeild in consequence of occasional attacks of pain. The lens and vitreous are almost entirely wanting. The choroid and retina are *in situ*. They present numerous black pigment spots, very much resembling those of retinitis pigmentosa.

Microscopical examination shows that the black spots are due to an increase in the pigment on the inner choroidal surface. It is extremely black and irregular, being in some places entirely absent. At one point a small dense fibrous and bony plate is seen imbedded in the pigment, and near it, but in the centre of the choroid itself, we find a similar, though much smaller plate. There are no inflammatory cells in the choroid. Its lamina vitrea is not thickened at any point. The ciliary body bears a pigmented membrane on its internal surface.

No. 84. Mary Ann Kelly, æt. 24. Her *left* eye is blind from ophthalmia neonatorum. She has occasional pain in it. Its tension is normal.

The leucomatous cornea presents a minute fistulous opening. The iris adheres to its posterior surface as a pigmented web. There is no lens. The optic disc is white.

The choroid and retina are *in situ;* the latter is mottled with dark pigment, giving an appearance somewhat like that of retinitis pigmentosa.

*Microscopical Examination.*—The retinal pigment epithelium

stratum is either entirely absent, in which case the outermost retinal layers are also wanting and the retina and choroid cohere, or the pigment is irregular, unduly abundant, and black. A small plate of bone is found close to the optic disc. It is difficult to be satisfied as to its exact position; but there is some evidence that the unthickened lamina vitrea of the choroid is on its external surface. It is overlaid on its inner surface by a fibrous stratum, apparently the remains of that in which the bone was developed. At other places pieces of bone are found enclosed in the very midst of the hypertrophied pigment epithelium layer.

No. 347. Thomas Ridgley, æt. 12, under care of Mr. Lawson. His *right* eye was lost soon after birth by perforating ulcer of the cornea, consequent on ophthalmia neonatorum. Its tension is much increased at the time of excision.

*Microscopical Examination.*—The relations between retina and choroid are almost exactly the same as in the previous specimen, except that the position of the bone *in* the hypertrophied pigment epithelium layer is more clearly apparent here, and that several large colloid-looking masses occupy the same position. There is a tough pigmented membrane on the inner face of the ciliary body.

No. 34. Sarah Wine, æt. 13. *Left* eye is large and staphylomatous in front, in consequence of an old perforating ulcer, due to ophthalmia neonatorum. There is a small, but rather thick plate of bone *in* the choroid near the optic disc. It occupies the greater part of the thickness of that membrane, the chorio-capillaris only remaining uninvolved. The latter is occupied by a considerable number of inflammatory cells. The lamina vitrea over it is unthickened. Its pigment epithelium is increased, irregular, and unduly black in colour. In some places it extends into the external layer of the subjacent retina. The ciliary body bears on its inner aspect a thin layer of new formation.

No. 335. Edmund M., æt. 20, under care of Mr. Couper. His left eye is shrunken in consequence of a spontaneous inflammation, apparently an iritis, four years ago, which affected the right eye also three or four months later. He has every appear-

ance of congenital syphilis, and his family history is consistent with this. Between the retina and choroid is a layer of dense fibrous tissue, much thicker than either. It is sparsely nucleated, and contains embedded in it at one point a bony mass of considerable size.

The choroid contains more nuclei than normal. Its vitreous lamina is unthickened and much undulated. The pigment on its internal surface may be very black, irregular, and much thickened, or it may be wanting.

No. 776. Jane R., æt. 60. *Right* eye excised on account of corneal staphyloma and glaucoma, secondary to corneal ulcer ten months ago. It has been occasionally painful. The retina and choroid are *in situ*. On the internal surface of the latter are many elevations, mostly small, but some as much as ·08 mm. in height. Most, when viewed on the flat, are darker in colour than the surrounding choroid. Some have an uncoloured centre, but are surrounded by a ring of dark pigment; others are entirely unpigmented.

Microscopic sections show that the dark raised spots are due to a new formation, either entirely structureless or containing a few nuclei, between the hypertrophied pigment epithelium and the unaltered vitreous lamina. It would appear as if the earliest condition of the new formation were that in which the structureless matrix contains a few nuclei, and is covered by darkened pigment epithelium, and that the entirely structureless form with diminishing covering pigment were a product of retrograde change.

The choroid shows some increase of its nuclei. These are, however, distributed most abundantly in its middle layers, and not in any special relation to the exudations on the inner surface of its vitreous lamina.

No. 695. Margaret H., æt. 25, under care of Mr. Streatfeild. Excision of the *right* eye, which was needled eight years ago on account of cataract. Now, it has no perception of light, and is, moreover, occasionally painful. The *left* eye has lately become irritable. In the *right* the retina is detached; the choroid *in situ*. On the internal surface of the latter we see many small elevations, the largest being ·07 mm. in height. They are

on the internal surface of the vitreous lamina, and appear to consist, at all events in larger part, of enlarged amyloid-looking cells of the pigment epithelium layer. Such separate cells are abundantly seen over the internal choroidal surface in the form of minute isolated nodules, diam. about ·01. Each has a large nucleus, which stains with the hæmatoxylin a little more deeply than the substance of the cell itself, to which this reagent gives only a slight diffuse blue coloration. The pigment granules are scattered over the surface of the cell, and are much less dark than normal.

No. 329. Margaret P., æt. 21. *Right* eye excised by Mr. Lawson, on account of its causing sympathetic irritation.

It was injured with a fork eight years ago. The retina is detached and partly contracted; the choroid has some minute dark elevations on its internal surface. These are caused by the presence of structureless material between the unthickened vitreous lamina and the pigment epithelium. It is clearly distinct from both. The epithelial cells overlying it are enlarged and very dark. Cells of the epithelial pigment layer at other points may be enlarged and degenerated, so as to form spheres projecting beyond the level of their darker-coloured neighbours. The nuclei of the choroid are more numerous than normal, but their distribution bears no particular relation to the presence of the colloid formations.

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Fig. 1.—Microscopical section of the choroid of No. 636, to show the thick fibrous layer in the external part of the choroid. The choroid is above, and a part of the sclerotic is visible below. The lower gap (on the left) is between the fibrous layer and sclerotic; the uppermost (on the right) is in the fibrous layer itself.

Fig. 2.—Section showing a part of the sclerotic, choroid, and detached retina of No. 63. A *colloid body* which contains a few nuclei, is continuous at its base, with a bony plate, which is internal to the unaltered lamina vitrea. The very considerable gap which exists between the choroid (above) and the sclerotic (below) is due to an accident in the preparation.

Fig. 3.—Section from No. 782, showing the bony network which nearly fills up the cavity of the shrunken eyeball. The choroid is the narrow dark band at the lower edge of the specimen. Between it and the bone are the peculiar rounded bodies described in the text.

*Fig. 1.*
× 35.

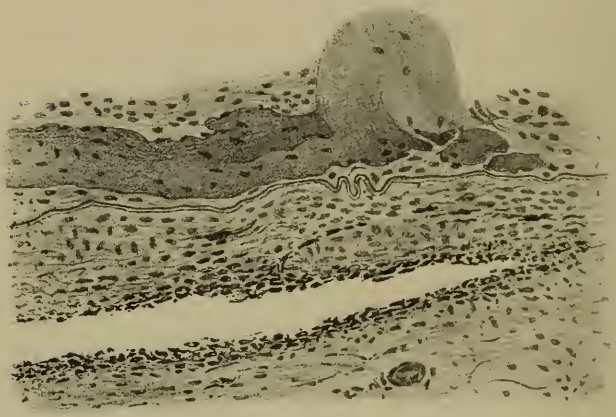

*Fig. 2.*
× 85.

*Fig. 3.*
× 16½.

## ON CHOROIDAL NEW FORMATIONS.

By W. A. Brailey, M.D., and Dr. Gama Lobo.

Choroidal New Formations may be bony, fibrous, or structureless, or they may present these various conditions in combination. They may be within the choroid, but are more commonly seen on its external surface.

One of the most striking of these consists in the development of a highly nucleated fibrous stratum between the bulk of the choroid and the sclera (*see* Plate, fig. 1). The newly-formed tissue, which looks to the naked eye like a somewhat thinner repetition of the sclera, probably represents the vastly thickened Lamina Suprachoroidea. It is separated from the sclera by a thin pigmented layer, the lamina fusca, and it has on its inner surface the rest of the choroid in the form of a pigmented layer, which contains many dilated thin-walled blood-vessels. It is more highly nucleated than the sclera, and the general arrangement of its component fibres is parallel to its surfaces, whereas the more distinctly differentiated scleral fibres have in these cases, as indeed is generally the case in shrinking eyes, a much undulated arrangement.

The Tunica Vasculosa cannot be traced with certainty as a distinct layer. It is most probable that the bulk of it is absorbed into this fibrous tissue stratum, so that the latter will represent not only the lam. supra-choroidea but also part of the tunica vasculosa.

The cavities by which this fibrous layer is traversed are very large. Indeed they give in section the appearance of cysts. Their continuity and their blood-like contents support the view that they are dilated blood-vessels. They have, however, no special walls of their own, but appear to be simply channelled in the highly-nucleated tissue which they

traverse in a direction parallel to its surface. In certain specimens a considerable cavity, which appears to represent the peri-choroidal lymph space, exists on the external surface of this layer (*see* Case No. 736).

The other new formations which attract most attention are the Osseous, either in the choroid or on its inner surface, and the so-called Colloid bodies which occupy the latter position.

But the two formations are intimately related, for it is by no means uncommon to find colloid nodules and bone associated in the same specimen. And we may even find bone corpuscles in such nodules or a plate of undoubted bony tissue on the lamina vitrea in immediate relation with them.

Bone is rare *in* the choroid in comparison with its occurrence on the inner choroidal surface, *i.e.*, on the retinal aspect of that conspicuous, delicate, homogeneous-looking lamina which, in ordinary microscopical sections, is seen to limit the choroid internally, and which we shall designate in this communication the Lamina Vitrea.

When bone is found within the choroid, it usually occupies the chorio-capillaris. In some cases it attains a much greater thickness than the normal chorio-capillaris, and then the remaining choroidal layers are thinner than normal, more pigmented and, as it were, compressed together. The formation of bone in the choroid does not proceed immediately from the lamina vitrea. Indeed, in many instances, there is a distinct interval, containing however many elongated nucleated cells between the latter and the newly formed bone. This may be the case even where bone consists on both aspects of the lamina vitrea.

Bone developed on the inner surface of the lamina vitrea may lie in the midst of the proliferated pigment epithelial layer. Such cases are not uncommon in wounds and perforated ulcers of long standing. In such cases the origin of the bone from the proliferated pigment epithelium is beyond doubt. And we may say the same with regard to the hard

nodules or bony plates which are sometimes found in or on the external layers of the detached retina.

In other instances one or more bony plates lie on the inner choroidal surface in the midst of a vascular highly-nucleated tissue. In these, the origin of the bone from the pigment epithelium is open to question, as all traces of the pigment cells may be wanting.

The several imperfect layers of which the thicker masses of bone are composed have, in section, the appearance of irregular anastomosing rods, the interspaces of which are occupied by an extremely delicate, moderately vascular connective tissue. One large, extremely thin-walled blood-vessel, apparently a dilated empty capillary, is usually conspicuous in the centre of each interspace. A comparatively thin, dense, sparsely nucleated, almost non-vascular fibrous layer usually terminates the new formation on its retinal aspect.

The newly-formed fibrous and osseous tissue thus constituted may form abrupt elevations on the inner choroidal surface, especially round the optic nerve entrance (*see* Case No. 762), or it may be more extended and attain an extreme thickness (*see* Plate, fig. 3), in which case there remains for the detached retina but a small cavity in the centre of the eyeball.

A delicate lamina vitrea can usually be traced as separating such formations from the comparatively thin and more pigmented choroid. The deepest layers of the new formation contain certain rounded* or elongated bodies, which, in their size and arrangement, remind us of the immediately adjacent layers or rods of bone (*see* Case No. 782, and Plate, fig. 3). But each is made of a more or less thick almost structureless wall enclosing a central yellowish granular substance. We lack evidence of the exact nature of these bodies. In many respects they resemble obstructed blood-vessels, whose walls are thickened and degenerated. Globes of similar yellowish

* Meyer, A. f. O., xxiii, 4, figures in a similar position certain rounded bodies which appear however to have the usual structure of so-called colloid bodies.

matter occupy in rare cases some of the interspaces of the bony network, and, more rarely still, similar globes, each enclosed in a hyaline ring, form projections on the retinal surface of the newly-formed bony and fibrous layer (*see* No. 730). Such may be either pedunculated or free lying. The latter condition may be explained by supposing that the section-knife has not passed through the peduncle.

In other cases, where the new formation takes the form of nodules on the retinal aspect of the lamina vitrea, these may be entirely or partially bony. Thus, we have a transition between the osseous formations and the so-called colloid bodies.

Sometimes these nodules are extremely large, even 1·5 mm. in height (*see* No. 566), and bands lying on the choroid may connect neighbouring ones.

Their structure is fibrous, fibro-cartilaginous, or osseous, the former predominating in their superficial layers, and the latter being more conspicuous towards their deeper part.

They have very commonly a central ill-defined cavity in which is yellowish granular matter, intermixed sometimes with colourless acicular crystals, the whole seeming to denote the previous existence of a vascular supply. Such bodies remind us in some degree of the yellowish granular spheres just described as existing within the new-formed osseous tissue or on its internal surface.

Other nodules, though still of fair size (·25 mm. in some instances), are more colloid like. For they are very commonly structureless in their superficial part, while their deeper part may contain well-developed bone corpuscles (*see* Plate, fig. 2), and do not contain any central cavity. They may rest directly on the unaltered lamina vitrea, or they may be separated from it by some nucleated fibrous tissue. In either case there may be a similar nucleated or bony tissue beneath the lamina vitrea.

It would appear that bone is never developed in the lamina supra-choroidea, or even in the tunica vasculosa of the choroid.

Besides the large bodies just described as existing on the inner choroidal surface, we have the smaller nodular elevations so long known as colloid deposits.

Some of them are distinctly composed of enlarged and but slightly pigmented cells of the pigment epithelial layer. One such degenerated cell may form a small projection, but more commonly a group of three or four forms a larger prominence. But the real colloid bodies are composed of a structureless basis substance between the lamina vitrea and its pigment epithelium, which may contain, especially in its deeper part, a few large oval feebly-staining nuclei, the whole having in this case somewhat the appearance of a so-called giant-cell; or the nodule may be calcified by the deposit of earthy salts in it.

It is extremely probable that these three conditions, the nucleated, the structureless, and the calcified, are successive stages. The overlying pigment epithelium presents in its appearance an associated variation. In one stage, probably the earliest or nucleated, its cells are enlarged and unduly black, so that the nodule, when viewed on the flat, is darker in colour than the surrounding choroid.

Or the pigment cells covering the centre may have vanished, leaving the colloid nodule set in a dark ring. When calcified, the nodules are entirely free from pigment cells, and constitute numerous small white pearly elevations. In connection with these changes, *see* Pagenstecher and Genth's admirable Atlas, Plate XVIII, fig. 1.

Such new formations, though they are said to be developed in senile eyes as a purely degenerative change, appear in the majority of cases to be the results of inflammation. Some of them, especially those which attain a considerable size, stain deeply with hæmatoxylin, and so uniformly that it is difficult to say whether corpuscular elements are present in them (*see* Nos. 248 and 63). In others the staining capacity is restricted to the central and deeper part.

These nodules have thus a position and origin different from the bony plates or nodules described as developed in the

midst of the hypertrophied pigment epithelium. Perhaps they are derived from that stratum of the lamina vitrea described as existing between the well-defined layer (to which we have in this paper restricted the term lamina vitrea) and the retinal pigment epithelium (Sattler, A. f. O. xxii, 2).

It will be gathered that we attach but little importance to the lamina vitrea as a source of these new formations. Indeed, in most cases it can be traced as a distinct layer, unaltered in structure, though much undulated. In rare cases, however, a thicker membrane, which appears to be continuous with the lamina vitrea, can be traced on to the pedicle of the detached retina in the immediate neighbourhood of the optic disc. It may also be studded with nodules which have every appearance of being thickenings of it. Perhaps examinations at an earlier stage may throw light on such apparently exceptional conditions.

The contributions of Wedl,* Iwanoff,† and Meyer‡ to the anatomy of colloid, are well known, as also the researches of Knapp§ and Goldzieher‖ on Osseous Formations (*see* also Becker's Atlas, Plates XXIII and XXIV).

Having discussed the nature and position of these new formations, a few words as to their etiology may not be out of place.

With regard to the first-described condition, the thickening of the lamina supra-choroidea, few cases have come under my observation. In one, the best marked, there was a clear history of necrosis of the cornea, the result of gonorrhœal ophthalmia. A condition somewhat similar in appearance is common as a result of the Choroiditis Maligna of sympathetic disease. The two, however, are for the most part clearly distinguished microscopically, for in the latter disease

* Atlas der path. Hist. Leipzig, 1860.
† Graefe and Saemisch, Handbook, vol. iv.
‡ A. f. O., vol. xxiii, 4.
§ A. f. O., vol. ii.
‖ A. f. O., vol. ix, 3.

the newly-formed tough, greyish, translucent fibrous tissue between Choroid and Sclerotic is much thicker and less dense. It is not at all improbable that the one may be the ultimate state of the other. If so, we must admit that choroiditis maligna, however typical its microscopical characters, does not always give rise to inflammations in the opposite eye.

In the cases above reported, I cannot fail to regard the colloid bodies on the Choroid as the results of inflammation. For there is in each of them a history of previous injury or disease, accompanied by pain and vascular injection.

I have no experience of the non-inflammatory senile colloid which has been described. It is rare, however, under any circumstances, to find exudation, which we can demonstrate to be inflammatory on the inner choroidal surface. On the contrary, exudations are conspicuously absent in Choroiditis Maligna, in Suppurative Hyalitis, or even in Suppurative Choroiditis, unless indeed the lamina vitrea chance to have been perforated. I am inclined to think, however, that they may be developed at a later stage of these choroidal inflammations, in which case they would be rather degenerative than acutely inflammatory. Their microscopic appearance does not indicate an acute stage of inflammation, for their nuclear elements are, as a rule, neither abundant nor deeply staining. In some cases, however, where old lost eyes have become suddenly painful, I have found deeply staining nodular elevations of considerable size on the lamina vitrea. But the microscopic characters of the accompanying choroidal inflammation were neither extensive nor of any well-marked type.

It is probable that the forms of inflammation which produce bone on the choroid are those which give rise to colloid deposits.

Inflammations accompanied by proliferation of the retinal pigment epithelium, are those which in many cases distinctly give rise to bone on the inner surface of the choroid. Wounds and perforating corneal ulcers are not infrequently followed by an inflammation possessing this character. In such cases

the general appearance of the fundus is very like that of Retinitis Pigmentosa. The Choroiditis sometimes met with in syphilis and also in myopia, may also present this character, and thus it comes that bone may be developed in consequence of these diseases. Such forms of Choroiditis are liable to be accompanied by a Cyclitis of a similar character. But the pigmented fibrous deposits on the inner surface of the ciliary body are rarely ossified. We find, however, an admirable example of localised ossification in No. 821. And as the choroidal changes may be diffuse or localised in spots, so we have spots or larger areas of fibrous and pigmented new formation on the inner surface of the ciliary body. Such localised areas are usually crescent-shaped, their long axis being parallel to the ciliary circle, and the subjacent ciliary body is atrophic, so that they project much less into the vitreous chamber than would be expected from their thickness (*see* No. 821).

Such forms of Cyclitis are, like the parallel form of Choroiditis, the results of injuries or of spontaneous inflammations which may possibly be connected with syphilis or myopia.

Calcifications within the lens capsule appear to be due to the same cause as osseous and colloid formations on the lamina vitrea, and the same may be said of the development of bone *in* the choroid.

# ON THE MEANING OF THE WORDS "NYCTALOPIA" AND "HEMERALOPIA" AS DISCLOSED BY AN EXAMINATION OF THE DISEASES DESCRIBED UNDER THESE TERMS BY THE ANCIENT AND MODERN MEDICAL AUTHORS.

By John Tweedy, F.R.C.S.,

*Professor of Ophthalmic Medicine and Surgery in University College, and Assistant Ophthalmic Surgeon at University College Hospital; Assistant Surgeon at the Royal London Ophthalmic Hospital.*

Whatever view we may take of the nature of logic, whether, with Archbishop Whately, we regard it as entirely conversant about language; or, with Sir William Hamilton and Dean Mansel, as concerned with the acts and states of mind indicated by the words we use; or, with J. S. Mill, as dealing with the things concerning which we argue; or, lastly, with Mr. Stanley Jevons, as in a certain sense embracing all three, namely, language, thought, and objects;[1] we must admit that language is one of the principal instruments of thought, and that any imperfection in it, or in the mode of employing it, is liable to confuse and impede the process of thought, and to destroy all ground of confidence in the result.[2] Unless our language be at once accurate, precise, and unequivocal, it cannot be trusted as an instrument either of record or of communication.

In treating of the connexion between words and ideas, and of the fallacies, errors, and disputes arising from uncertain or mistaken significations of words, Locke says, "I am apt to imagine that, were the imperfections of language, as the instrument of knowledge, more thoroughly weighed, a great many controversies that make such a noise in the world would of themselves cease; and the way to knowledge, and perhaps peace too, lie a great deal opener than it does."[3] This is especially true of the language of science, where words are

[1] Jevons: "Elementary Lessons in Logic," 4th ed., p. 10.

[2] J. S. Mill: "A System of Logic," 7th ed., vol. i, p. 17.

[3] Locke: "An Essay concerning the Human Understanding," book iii, c. ix, sec. 21.

not only terms but definitions also. Hobbes defined *a name* as "a word taken at pleasure to serve for a mark, which may raise in our mind a thought like to some thought we had before, and which being pronounced to others, may be to them a sign of what thought the speaker had before in his mind."[1] The convenience and necessity of preserving the signification of words untainted is obvious. If I write the word "conjunctivitis," or "iritis," or "cataract," or "myopia," the reader at once forms a sufficiently intelligible idea of what I mean. So also with "mydriasis:" the mention of this word forthwith calls up the idea of a fixed dilated pupil, though here and there, perhaps, might be found one who, entrenching himself behind the sole authority of Aretæus,[2] would, pedantically, declare that the word called up in his mind the idea of "a pupil contracted to a small size." But what idea do I provoke when I employ the word "nyctalopia?" Nine persons out of ten, if they had the courage and confidence to avow it, would reply that they were not quite clear whether the term meant *night-blindness* or *night-sight:* though if pressed they would pronounce in favour of the latter. Nor would the answer seem unreasonable if we remembered that in all the best known published texts of Hippocrates, and in all the translations of this author, and in nearly every book written on Ophthalmology during the last century and a-half, the term "nyctalopia" is employed to denote the disease in which a person can see well by night and not by day. It is in this sense the word *νυκτάλωψ* is defined in the latest (6th) edition of Liddell and Scott's Greek-English Lexicon, on the one hand, and in the most recent and important treatise on modern Ophthalmology, on the other,[3] as well as in nearly every known scientific and medical lexicon, including that of

[1] "Computation or Logic," c. ii; cited by J. S. Mill, *op. cit.*, vol. i, p. 23; and by Jevons, *op. cit.*, p. 17.

[2] "De Sig. et Caus. Diut. Morb.," lib. i, c. vii; also "The Extant Works of Aretæus, the Cappadocian," trans. and edited by F. Adams, LL.D. Syden. Soc.: London, 1856; pp. 65 and 307.

[3] Graefe und Saemisch: "Handbuch der Gesammt. Augenheilkunde."

Blancard,[1] Kühn,[2] and the scholarly production of Littré and Robin.[3]

In spite of this remarkable consensus of opinion among modern authorities regarding the meaning of the word nyctalopia, it can be shown that according to the authority of the best classical writers, and according to the etymological construction of the word, the real meaning of the term nyctalopia is *night-blindness;* and consequently, that the meaning of the opposite term hemeralopia is *day-blindness,* and not *night-blindness,* as modern writers assert.

I have already mentioned the circumstances that led me to revive the discussion on the meaning of the words nyctalopia and hemeralopia.[4] The last number of these Reports contains a letter kindly and promptly written at my suggestion by Dr. W. A. Greenhill, the learned editor of Theophilus, of Celsus, and of Sydenham, and giving the results of a critical examination of the use of these words in the ancient Greek and Latin authors. The evidence adduced by Dr. Greenhill is conclusive. Henceforth, no one can defend the use of the word hemeralopia for *night*-blindness, and nyctalopia for *day*-blindness on philological or literary grounds. Dr. Greenhill has explained how the confusion arose. According to the ordinary texts of the Hippocratic writings, "nyctalopes" are said to be "those who see at night," whereas, for the reasons stated at p. 287, note 3, the original definition almost certainly was "those who do *not* see at night." This conjecture Dr. Greenhill defends with great ingenuity and learning. His position may, I believe, be further strengthened by comparing the clinical and pathological affinities of the nyctalopic affection described in the Hippocratic writings and the night-blindness of subsequent authors.

[1] "The Physical Dictionary :" London, 1693.

[2] "Lexicon Medicum :" Lipsiæ, 1832, vol. ii, pp. 10–18.

[3] "Dictionnaire de Médicine :" 14th ed. Paris, 1878.

[4] Prefatory Note to Dr. Greenhill's letter "On the meaning of the words 'Nyctalopia' and 'Hemeralopia,'" published in the last number of these Reports, p. 284.

It is this latter task I wish more particularly to attempt in this article.[1]

Many reasons might be assigned for believing the true meaning of the word nyctalopia to be night-*blindness*, and not night-*sight*. The principal ones are seven in number, and may be briefly expressed thus:—

1. All the ancient Greek medical writers, with the questionable exception of Hippocrates, and nearly all the Latin medical authors down to the end of the fourteenth century, as well as all other authors down to the middle of the seventeenth century, who define the term nyctalopia, use it in the sense of night-blindness.

2. As a *symptom* night-blindness is more frequent, more obtrusive, and more characteristic than night-sight. Indeed most, if not all, of the alleged cases of the latter are instances of dread of light,—photophobia, or heliophobia (Demours),—due to inflammation of some of the tunics of the eyeball, rather than of blindness by day and sight by night.

3. As an *amaurotic state or condition* night-blindness, whether it occur sporadically, or endemically, or epidemically, is comparatively frequent, and has been recognized in all ages, while the opposite condition of true night-sight is extremely rare, if not altogether unknown.

4. Even as regards the nyctalopic affections mentioned in the Hippocratic writings, there is almost conclusive evidence that they are essentially the same as those described by Aristotle and other writers as night-blindness. Most of the associated and predisposing conditions are the same, and many of the alleged concomitants are clearly those of night-blindness.

[1] It should perhaps be mentioned that this article was originally written to appear in conjunction with Dr. Greenhill's letter. It was, however, found on comparing them in print, that they travelled over much of the same ground, though for different purposes. It was, therefore, decided to publish Dr. Greenhill's letter first and independently, in order to keep the philological and the pathological arguments distinct and separate. In the interval I have taken the opportunity to revise and extend some portions of my article.

5. There is excellent manuscript authority that the writer of the second book of the Hippocratic treatise, named Prorrhetics, did actually state that persons who do *not* see at night are called nyctalopes.

6. If the term nyctalopia do not denote night-blindness, then we are compelled to conclude either that Hippocrates failed to recognize the affection; or, if he did recognize it, he did not deem it necessary to describe it or even to mention its special name.[1]

7. If the term nyctalopia originally meant night-*sight*, then we must conceive the philological absurdity of describing by a word having reference to night (*νύξ*), a pathological state or condition characterized by an inability to see during the *day*.

If these reasons be considered more closely, their force may perhaps become more apparent.

(1). The term nyctalopia is used to denote night blindness by every one of the best Greek medical writers from Aristotle to Joannes Actuarius. It is used in this sense also by Pliny in the first century; by Paré, Guillemeau, Schenk, and Forest in the sixteenth; and by Riolan, Ettmuller, and others in the seventeenth, century.

A glance at Table I will show that of all the authors whose writings are quoted, to the middle of the seventeenth century, only one, namely Hippocrates,[2] may be cited as using the term nyctalopia in the sense of *night-sight*. It is true that some confusion of meaning seems to have existed in the time of Aëtius[3]

[1] It should be remembered that the term *hemeralopia*, now used to denote night-blindness, was not known to the ancient authors; *see* Dr. Greenhill's letter, p. 284.

[2] "Prorrhet.," ii.

[3] Tetra. ii, serm. iii, c. xlvi. Though Aëtius describes nyctalopes as persons who see well by day and nothing at all by night, he also states that it happens to some (nyctalopes?) that they see *better* by night than by day, adding that this is rare. It may be that in the latter instance Aëtius is really referring not to nyctalopes but to hemeralopes, without using the term. The passage is, however, taken in the sense of nyctalopes, by the Paris editors of Stephanus' "Thesaurus;" by Foësius, in his edition of Hippocrates, sec. ii, p. 91 (Francof., 1595), and by Plempius, "Ophthalmographia."

in the fifth century, but Plempius,[1] in the seventeenth century, is one of the first writers who distinctly states that the term nyctalopia bears the double signification of night-sight (Hippocrates) and of night-blindness (Aëtius, Actuarius, and Paulus Aegineta). Plempius himself evidently inclines to the opinion that the former is the more correct, since he says, "Unde et Celsus non recte *Hippocratis* sententiam nocturnæ cæcitati accommodat."[2] Celsus,[3] it may be remarked, described night-blindness under the term "imbecillitas oculorum."

In the accompanying Table (I) only those authors are named who actually define the term nyctalopia or its cognates.

Table I.

NYCTALOPIA.

| Night-Sight. | Night-Blindness. |
|---|---|
| Hippocrates (?)[4] | Aristotle.[5]<br>Pliny.[6]<br>Galen.[7]<br>Oribasius.[8]<br>Aëtius.[9]<br>Alexander Trallianus.[10]<br>Paulus Aegineta.[11]<br>Joannes Actuarius.[12] |

[1] "Ophthalmographia," 3rd ed.: Louvanii, 1659; lib. v, c. xxvi, p. 232*b*.

[2] *Op. cit.*, p. 234*a*.

[3] Lib. vi, c. vi., sec. 38.

[4] "Prorrhet.," ii.

[5] "De Generat. Animal.," lib. v, c. i.

[6] "Hist. Nat.," lib. viii, c. lxxvii; lib. xxviii, c. xlvii.

[7] "Obselat. voc. Hippoc. Explanat.," in Froben's Latin ed., Basil, 1561, vol. ix, p. 149; or Cratandar's Greek ed.: Basil, 1538, pars v, p. 714. *See also* Dr. Greenhill's letter, p. 285, note 4.

[8] "Synops.," viii, 46: "De loc. affect. curat. ad Eunap.," iv, 18.

[9] Tetra. ii, serm. iii. c. xlvi.

[10] "De Arte Medica," lib. ii, c. vi.

[11] "De Re Medica," lib. iii, c. xxii: "De Nyctalope."

[12] "Med. sive de Method. Medendi," lib. iv, c. xi; *ibid.*, lib. ii, c. vii, *νυκτάλωπας ἰατρινοὺς*.

NYCTALOPIA—*continued.*

| Night-Sight. | Night-Blindness. |
|---|---|
| (16*th Century*). | (16*th Century*). |
| | Paré.[1] |
| | Guillemeau.[2] |
| | Forestus.[3] |
| | Schenkius.[4] |
| (17*th Century*). | (17*th Century*). |
| | Riolan.[5] |
| | Hoefer.[6] |
| | Ettmuller.[7] |
| Plempius.[8] | Plempius (?)[8] |

It would therefore appear, that, with the possible exception of Hippocrates, and the unknown writer of the Galenic work, "Introductio, seu Medicus," no author of repute used the term nyctalopia to denote night-*sight* or day-*blindness*, before the seventeenth century. Even Plempius does not limit the term to night-sight; he rather speaks of two forms of nyctalopiasis, in one of which the patient cannot see well by night, in the other he cannot see well by day.

After the seventeenth century a very remarkable change occurs. The term nyctalopia loses the signification it has hitherto possessed, and gradually takes on that of night-sight, while the term hemeralopia is come into use to denote night-blindness. It is difficult to account for this change of meaning.

[1] "Œuvres Complètes d'Ambroise Paré," par J. F. Malgaigne: Paris, 1840, vol. ii, p. 415; liv. xv, c. v.

[2] "Traité des Maladies de l'Œil:" Paris, 1585, p. 27.

[3] "De Morbis Oculorum," 1591, or "Observat. et Curat. Medic.:" Francof, 1602, lib. xi, obs. xxxviii.

[4] "Observat. Med. Rar.," ed. by J. G. Schenkius: Francof., 1665, lib. i, p. 159.

[5] "A sure guide on the nearest and best way to Physick and Chyrurgery," trans. by N. Culpeper: Lond., 1671.

[6] "Hercules Medicus:" Noriberg, 1675, lib. i, c. viii.

[7] "Opera omnia:" Francof., 1688, "De Visus laesionibus," p. 475.

[8] "Ophthalmographia," 3rd ed.: Lovanii., 1659, p. 232.

The term hemeralopia (except in the "Introd. seu Med.") is seldom to be met with in medical writings before the eighteenth century, though it is evident from the descriptions given by Paré, and especially by Guillemeau, that the term was not unfamiliar in their time.

Paré (*circa* 1578) says: "*Acies nocturna:* quand on void mieux de nuit que de iour, et se peut dire *Hemeralopia* en grec, *Œil de chat* en françois;"[1] and Guillemeau, writing in 1585, speaks of "aveuglement de jour, dict en Grec *Ημεραλωπια.*"[2] Plempius, seventy-four years later, does not mention the term hemeralopia at all, though he discusses the problem "Cur nonnullorum oculi noctu sunt perspicaces, interdiu hebetiores?"[3]

With the eighteenth century, the term hemeralopia comes into frequent use, and almost invariably bears the meaning of night-blindness, instead of day-blindness as heretofore; while the meaning of the term nyctalopia, on the other hand, in its turn, is, as I have said, transferred from night-blindness to day-blindness. Mâitre-Jan as well as Bergen and Weise continue to employ nyctalopia as equivalent to night-blindness or day-sight, but Boerhaave, Guerin, and Sauvages use the word hemeralopia to denote this condition, while they apply the term nyctalopia to a confessedly rare condition of night-sight. The probable explanation of this extraordinary alteration, is that before the sixteenth century the works of Hippocrates were not accessible, and were consequently rarely consulted. Galen was the chief authority for the later Greeks and Romans, as well as for the Arabians; and though Hippocrates was held in high esteem by the ancient Greek and Latin physicians, he was not much studied during the Middle Ages. Galen had defined the Hippocratic term "nyctalopes" as those who are blind at night, and his definition was unquestioned till greater facilities occurred for

[1] *Loc. cit.*
[2] *Op. cit.*, p. 28.
[3] *Op. cit.*, lib. iv, prob. lviii, p. 177*b*.

consulting the Hippocratic treatises in the original.[1] In the sixteenth century Mercurialis (1588) and Foësius (1595) published their editions of the Hippocratic writings, and in the seventeenth century Vander Linden's edition appeared (1665). From this time more attention was given to the Hippocratic writings, and in the seventeenth and eighteenth centuries medical authors appealed to them in the original.

In all these editions the reading of the passage in the second book of the Prorrhetics is: "*Οἱ δὲ τῆς νυκτὸς ὁρῶντες, οὓς δὴ νυκτάλωπας καλέομεν. κ.τ.λ.*" "Those who see at night, whom we call nyctalopes, etc." Hence those who consulted the Hippocratic writings would naturally conclude that they had discovered an error in the commonly accepted meaning of the term nyctalopia, and that the term really meant night-sight, and not night-blindness, as Pliny, Galen, Aëtius, Paulus Aegineta, and others had supposed.

This reading is still generally accepted as authentic. Even Littré[2] did not think it necessary to alter it, though he mentions that its accuracy had been impugned by Coray.[3] In some other quarters, however, the reading has been blindly accepted without any suspicion of doubt or difficulty. The late Sir William Lawrence, for example, was not content merely to accept the common reading of the Hippocratic text, but in order to justify the use of the word hemeralopia to denote night-blindness, actually states, "Hippocrates uses the term hemeralopia to denote night-blindness, and we may as well follow his example,"[4] whereas Hippocrates does not use the term hemeralopia at all.

An examination of Table II will show how completely writers have turned about since the seventeenth century.

[1] See further on this question Dr. Greenhill's articles on Galen and Hippocrates in Smith's "Dict. of Greek and Roman Biography and Mythology," vol. ii, pp. 210 and 435.

[2] "Œuvres Complètes d'Hippocrate," tome ix, p. 65, note 9.

[3] Traité d'Hippocrate, "Des Airs, des Eaux, et des Lieux:" Paris, 1800; vol. ii, p. 45, *et seq.*

[4] "A Treatise on Diseases of the Eye," 3rd ed.: London, 1844, p. 584.

TABLE II.

NIGHT-BLINDNESS.

| Nyctalopia. | Hemeralopia. |
|---|---|
| (18*th Century*). | (18*th Century*). |
| Maître-Jan.[1] | Boerhaave.[4] |
| Bergen and Weise.[2] | Guerin.[5] |
| | Sauvages.[6] |
| (19*th Century*). | (19*th Century*). |
| Grant[3] | De Wenzel.[7] |
| | Bampfield.[8] |
| | Demours.[9] |
| | Simpson.[10] |
| | Mason Good.[11] |
| | Scarpa.[12] |
| | De la Rue.[13] |
| | Wardrop.[14] |

[1] "Traité des Maladies de l'Œil:" Troyes, 1707; p. 271.

[2] "De Nyctalopia seu Cæcitate Nocturna." Haller, "Disput. ad Morb.," p. 359; Franc., 1754.

[3] "Cyclop. of Pract. Med.;" edited by Forbes, Tweedie, and Conolly: London, 1834; Art. "Nyctalopia;" vol. iii, p. 183.

[4] "De Morbis Oculorum;" ed. Gotting: Gottingæ, 1750; p. 159.

[5] "Essai sur les Maladies des Yeux:" Lyon, 1769, p. 289.

[6] "Nosologia Methodica Oculorum." Selected and translated by George Wallis, M.D: London, 1785, p. 190.

[7] "Manuel de l'Oculiste:" Paris, 1808; vol. i, p. 345.

[8] "A Practical Essay on Hemeralopia, or Night-blindness," read Dec. 7th, 1812: "Med. Chir., Trans.": London, 1814; vol. v, p. 32, *et seq.*

[9] "Traité des Maladies des Yeux:" Paris, 1818; vol. i, p. 423.

[10] "Observations on Hemeralopia or Nocturnal Blindness:" Glasgow, 1819.

[11] "A Physiological System of Nosology:" London, 1820; p. lxix, and pp. 300–3. Also "The Study of Medicine," 3rd ed.: London, 1829, vol. iv, pp. 200–1.

[12] "Saggio di Osservazioni e d'esperienze sulle Principali Malattie degli Occhi:" Pavia, 1801, p. 251; or the French translation by MM. Fournier-Pescay et Bégin: Paris, 1821; vol. ii, p. 318; or the English translation by James Briggs, 2nd ed., p. 471: London, 1818.

[13] "Cours Complet des Maladies des Yeux;" 2nd ed.: Paris, 1823; p. 370.

[14] "The Morbid Anatomy of the Eye;" 2nd ed.: London, 1834; vol. ii, p. 205.

NIGHT BLINDNESS—*continued.*

| Nyctalopia. | Hemeralopia. |
|---|---|
| (19*th Century*)—continued. | (19*th Century*)—continued. |
| Copland.[1] | Middlemore.[3] |
| Power.[2] | Tyrrell.[4] |
| | Hull.[5] |
| | Vidal (de Cassis).[6] |
| | Chelius.[7] |
| | Lawrence.[8] |
| | Desmarres.[9] |
| | Forbes.[10] |
| | Fabre.[11] |
| | Mayne.[12] |
| | Deval.[13] |
| | Costello.[14] |
| | Stellway von Carion.[15] |
| | Soelberg Wells. |
| | Seitz and Zehender. |
| | De Wecker. |
| | Galezowski. |
| | Tetzer. |
| | Schweigger. |
| | Macnamara. |
| | Lawson.[16] |

[1] "A Dictionary of Practical Medicine:" London, 1844.

[2] "Diseases of the Eye:" London, 1868; p. 453.

[3] "A Treatise on the Diseases of the Eye, etc.:" London, 1835; vol. ii, p. 225.

[4] "A Practical Work on the Diseases of the Eye:" London, 1840; vol. ii, p. 283.

[5] "Cursory Notes on the Morbid Eye:" London, 1840; p. 210, *et seq.*

[6] "Traité de Pathologie externe," etc.: Paris, 1840; vol. iii, p. 188.

[7] "Handbuch der Augenheilkunde:" Stuttgart, 1843; vol. i, p. 358.

[8] "A Treatise on Diseases of the Eye;" 3rd ed.: London, 1844; p. 584.

[9] "Traité des Maladies des Yeux: Paris, 1847; p. 696.

[10] "Cyclop. of Practical Medicine;" vol. iv: "Select Medical Bibliography;" p. 124.

[11] "Bibliothèque du Médicin-Practicien:" Paris, 1849; vol. x, p. 100.

[12] "An Expository Lexicon:" London, 1860.

[13] "Traité de l'Amaurose:" Paris, 1851, p. 294.

[14] "Cyclop. of Practical Surgery:" London, 1861.

[15] It is not necessary to give detailed references to the writings of the following authors, as their works are well known and in general use.

[16] "Diseases and Injuries of the Eye;" 3rd ed.: London. In the fourth edition, 1880, Mr. Lawson, at my suggestion, substituted the term nyctalopia for hemeralopia, to denote night-blindness.

NIGHT-BLINDNESS—*continued.*

| Nyctalopia. | Hemeralopia. |
|---|---|
| | (19*th Century*)—continued.<br>Bader.<br>Arlt.<br>Hersing.<br>*Et alii.* |

Had the classification adopted in Table I been continued, it would have been found that nearly every author of the eighteenth and nineteenth centuries had been cited as using nyctalopia in the sense of night-*sight*. As no useful purpose would have been served by continuing such a classification, I have, in the second table, adopted another plan, namely, to arrange the authors of the eighteenth and nineteenth centuries according as they use the word nyctalopia or hemeralopia to denote night-blindness.

The general agreement among modern authors in using hemeralopia to denote night-blindness is startling, and might be held to be a sufficient reason for abstaining from any attempt to disturb the acquired *status quo* or to recover the original synonym. Even Dr. Copland, who uses the word nyctalopia in the sense of night-blindness, does so on inadequate and unsatisfactory grounds; and Mr. Power confessedly merely copies, without criticism or comment, the definition given in Copland's dictionary. Consequently, we are compelled to fall back upon Dr. Grant as one of the very few authors of the nineteenth century who deliberately use the term nyctalopia in the sense of night-blindness. Even he does not advance any specific justification of such a choice; and the advantage of his decision is minimised by the course taken by Dr. Forbes, the principal editor of the "Cyclopædia of Practical Medicine," in ignoring Dr. Grant's opinion, and adopting the opposite meaning in the Select Medical Bibliography appended to the fourth volume of this work.

The results of this examination and comparison of ancient and modern writers may, therefore, be thus summarized:

Up to the end of the fifteenth century writers are almost unanimous in using the word nyctalopia to denote night-blindness. The latter half of the seventeenth century is a period of transition. From the beginning of the eighteenth century onwards writers use the word hemeralopia in the sense of night-blindness, and nyctalopia for day-blindness.

(2). As a *symptom* night-blindness is more frequent, more obtrusive, and more characteristic than night-sight. Every ophthalmic surgeon is familiar with night-blindness as a symptom of some diseases of the retina and of the choroid, and, in a lesser degree, of certain anomalies of refraction and accommodation; but no such symptom as blindness by day with sight by night ever occurs. In some cases of keratitis, of iritis, and of choroiditis, there is more or less photophobia, so that vision is somewhat better when the eyes are shaded or when the light is dull. Most albinoes too see better in a dim light; and whenever there is a small central opacity of the cornea or crystalline lens, vision is better in a dull than in a bright light, because then the pupil dilates, and rays of light pass through the clear margin of the cornea or lens. This is one of the forms of *visus nocturnus* of Boerhaave,[1] which he regards as identical with the nyctalopia of Hippocrates. The true nature of this form of night-sight is easily recognized, and in the strict sense of the term there is neither blindness by day nor sight by night. A similar criticism may apply to some of the instances referred to by Pliny, Plater, Rhodiginus, Plempius, Briggs, Boyle, Willis, Schenkius, Porterfield, and others, of persons possessing the power of seeing and even reading in the dusk or dark. So far as these accounts are trustworthy, they merely show that some persons may possess or acquire a degree of visual power enabling them to distinguish objects in an imperfect illumination. In the year 1867, Dr. Arlt,[2] of Vienna, described

[1] *Op. cit.*, pp. 137 and 162: Probably the high authority of Boerhaave was the chief factor in establishing the change of meaning of nyctalopia.

[2] "Der Bericht ueber die Augenklinik," 1867. See also American translation by Dr. Weightman: Philadelphia, 1868.

a form of retinitis which he designated *Retinitis nyktalopica*, in which, during the day, all objects appear to be covered by a silvery fog or haze, which is not noticeable at dusk. This form of retinitis does not, however, materially affect the present question.

(3). It is scarcely necessary to bring forward any special testimony as to the occurrence of night-blindness as an amaurotic affection. It is known to occur sporadically, or as an epidemic, or to prevail endemically in certain localities. The opposite condition of night-sight is probably unknown as an amaurotic condition.

It is a matter of common knowledge that night-blindness as an amaurotic affection is still prevalent in certain localities. On the other hand, it is doubtful whether any amaurotic condition is known in which the eyes are blind during the day, but possess the power of seeing at twilight or in the dark. Ramazzini[1] has alleged, that about the spring of the year he frequently met with young persons who were unable to see during the day, but who could see when night came on again. In about a month they recovered. These observations have not, however, been confirmed by other competent observers. Demours,[2] Middlemore,[3] Tyrrell,[4] Lawrence,[5] Mackenzie,[6] and most other authors, either deny the existence of such a disease, or assert it to be of extremely rare occurrence. The cases quoted by Mettrie,[7] Lassus,[8] Hillary,[9] Pye,[10] M. Guthrie,[11]

[1] "De Morbis Artificum," c. xxxviii; "Opera," p. 363: London, 1718.

[2] *Op. cit.*, vol. i, p. 432.

[3] *Op. cit.*, vol. ii, p. 233.

[4] *Op. cit.*, vol. ii, p. 283.

[5] *Op. cit.*, p. 589.

[6] "A Practical Treatise on Diseases of the Eye;" 4th ed.: London, 1854; p. 996.

[7] "Institutions de Médecine de Boerhaave, avec un Commentaire;" 2nd ed.: Paris, 1747; tome iv, pp. 431, 432.

[8] "Pathologie Chirurgicale," 1805–6; tome ii, p. 539, *et seq.*

[9] Quoted in Cooper's "Dictionary of Practical Surgery," Art. "Nyctalopia," vol. ii, p. 364.

[10] *Ibid.*

[11] "Duncan's Med. Commentaries;" vol xix, p. 290: Edinburgh, 1795.

Larrey,[1] Isbell,[2] and others will not enable us to form a category of diseases characterised by night-sight or day-blindness, in the same manner as we may form a category of diseases characterised by night-blindness. The predominating symptom in most, if not all the cases mentioned is, as I have already stated, dread of light, or photophobia. I have often seen persons engaged in out-door occupations who were thought to be suffering from day-blindness. They could see fairly well in the twilight, but were unable to get about during the day in bright sun-light. In every instance I found either a small vascular ulcer or other affection of the cornea, or some form of chronic conjunctivitis. The symptom was, therefore, photophobia, due to inflammation, and not amaurotic.

(4). A comparison of the meagre accounts given in Hippocrates of the condition of the persons designated "nyctalopes," and the fuller descriptions of night-blindness by later authors will, I believe, lead inevitably to the conclusion that the nyctalopes of Hippocrates are the same as the night-blind individuals of later authors; and, hence, that Hippocrates, or the writer of the second book of the Prorrhetics, intended to say that persons *not* seeing at night are called nyctalopes. This comparison of Hippocrates with the later authors applies not only to the definition of nyctalopia, but also to the description of the disease, to its course, its etiology, and its treatment.[3]

The predisposing and indisposing conditions, the concomitants, and other peculiarities of Hippocrates' nyctalopia, may be briefly enumerated as follows:—

(*a*) It chiefly occurs in children, and young adults.[4]

[1] "Mémoires de Chirurgie Militaire;" tome i, p. 6: 1812.

[2] "Edinburgh Med. and Surg. Journal;" vol. ix, p. 269: Edinburgh, 1813.

[3] It should be borne in mind that though Hippocrates is usually spoken of as the author of the works cited in this article, the best critics do not, for other reasons, believe that any of the treatises containing references to nyctalopia were really written by him.

[4] "Epid.," vi, sec. 7; "Prorrhet.," ii.

(*b*) Dark eyes, and eyes with small pupils are predisposed to it.[1]

(*c*) Married women and virgins whose menses are regular are not subject to it.[2]

(*d*) Eruptions appear about the ears on the seventh or eight day.[3]

(*e*) The formation of abscesses, and spontaneous diarrhœa are favourable prognostics.[4]

(*f*) The treatment consists of bleeding, purging, cupping, and the injection of ox's liver steeped in honey.[5]

(*g*) The disease is sometimes complicated by swelling of the gums, toothache, foul breath, swelling below the eyes, bleeding from the nose, ulcers on the legs, and enlarged spleen.[6]

All these circumstances and peculiarities are, separately or combinedly, mentioned by other authors as pertaining to night-blindness.[7]

*Age.*—It is commonly assumed that Hippocratic nyctalopia is a disease of childhood. M. Sichel, in the introduction to his translation of the treatise *Περὶ ὄψιος*, published in Littré's edition of Hippocrates,[8] contends that the disease designated nyctalopia, and described in the second book of the Prorrhetics, corresponds exactly with the scrofulous and some of the epidemic ophthalmiæ of children. This assumption is not justified by the text, *νέοι, ἢ παῖδες ἢ νεανίσκοι.* The term *νέοι* applies not only to children, but to *men at least as old as thirty*,[9] and the term *νεανίσκοι* certainly

[1] "Epid.," vi, sec. 7.

[2] "Prorrhet.," ii.

[3] "Epid.," iv.
"Prorrhet.," ii.

[5] "De Visu."

[6] "Prorrhet.," ii.

[7] It is not now the question whether these allusions are true or not. They are merely cited as illustrating the opinions of the various authors respecting night-blindness, and as showing that they ascribed to night-blindness the same qualities as those said to belong to nyctalopia.

[8] Tome ix, p. 122, *et seq.*

[9] *See* Liddell and Scott's Greek Lexicon, *νέος* and *νεανίσκος*.

includes men old enough to engage in battle, and to marry.[1]

The disease may, therefore, be said to occur not only in children (*παῖδες*), but also in young adults up to the age of forty. Moreover, the statement, presently to be discussed, that married women are not subject to the complaint, also implies that the disease is not exclusively one of childhood.

*Colour of the eyes, etc.*—Dark eyes and eyes with small pupils are said to be especially predisposed to Hippocratic nyctalopia.[2] Aristotle and other authors state these eyes show a similar predisposition to *night-blindness*.

In treating of some of the causes of the difference in colour of the eyes of different individuals, Aristotle remarks that it had been assumed, on insufficient grounds, that pale blue eyes (*γλαυκόμματα*) have more of fire, as Empedocles had said, and dark eyes (*μελανόμματα*) more of water than of fire; adding that it had been incorrectly stated that lack of moisture is the reason why blue eyes cannot see well by day, as want of fire is the reason why black eyes cannot see well by night.[3] In opposition to this view Aristotle asserts, with Democritus, that in every case vision is of water, and not of fire.[4] The eye is made of water, because this is transparent; and the amount of fluid (*τὸ ὑγρόν*) in the eye determines the colour of the eye. Eyes that have much moisture are dark, and those that have little are blue. This is the reason why blue eyes cannot see well by day, or black eyes by night; because blue eyes, on account of the scarcity of their moisture, are too much agitated by the light, whereas black eyes, from an excess of moisture, are not so easily set in motion.[5] Moreover, nocturnal light is very feeble, and does

[1] Herodt. iv, 72, 112, 114 and 115.

[2] "Epid.," vi, sec. vii, 1.

[3] "De Generat. Animal.," lib. v, c. i.

[4] *Ibid.*; and "De Sensu," c. ii.

[5] Aristotle's theory of vision was that visible objects set in motion a transparent medium between them and the eye, and that this motion is, in its turn, communicated to the sense-organ. "De Anima.," lib. ii, c. vii; "De Sensu," c. ii and iii; "De Generat. Animal.," lib. v, c. i.

not excite sufficiently the copious fluids of the dark eyes. Corresponding differences are, he continues, to be observed in the kind of diseases that affect blue eyes and dark eyes respectively. Cataract (*γλαυκωμα*) occurs in blue eyes, and "nyctalopia" in dark eyes. Cataract being a dryness of eyes, chiefly affects old people, but nyctalopia being due to a superabundance of moisture occurs in those who are younger (*τοῖς νεωτέροις*).[1] Similarly, Levinus Lemnius, though he does not use the term nyctalopia, says that dark eyes may be of such condition that they see acutely during the day, but badly and less clearly at night. (Noctu vero obtuse minusque acute videndi munere funguntur.)[2]

*The size of the pupils.*—Both Plempius[3] and Boerhaave[4] describe the connexion between night-blindness and narrowing of the pupil. Indeed, both these writers ascribe night-blindness to contraction and non-dilatability of the pupil rather than to thickening and opacity of the tunics and humours of the eye, as Aëtius had supposed;[5] "Quia in nyctalopia seu cæcitudine nocturna vesperi ægri non vident, sic tamen, ut die sat recte cernant; at qui crassas tunicas humoresque sortiti sunt, ne quidem die res probe intuentur."[6] According to Boerhaave the power of seeing at night is determined by the degree of dilatability of the pupil, and the reason why horses see better than men in the dark, is that horses have larger pupils, and hence more light can enter their eyes, but if the pupil do not dilate then they cannot see at night.[7]

*The Immunity of Women.*—Hippocrates states that married women and virgins who have the menstrual discharge

[1] Not necessarily young children, but *men of military age.* Thucyd., v, 50. *See* Liddell and Scott. *νεώτερος.*

[2] "De Miraculis Occult. Naturæ." Francof., 1604; lib. iv, c. vi, p. 375.

[3] *Op. cit.*, p. 233.

[4] *Op cit.*, p. 160.

[5] Tetr., ii; serm. iii, c. xlvi.

[6] Plempius, *loc. cit.*

[7] *Op. cit.*, p. 165.

regular are not subject to nyctalopia.[1] Celsus long after claimed for them a similar freedom from *night-blindness*:—"Præter hæc, imbecillitas oculorum est, ex qua quidam interdiu satis, noctu nihil cernunt; quod in fœminam bene respondentibus menstruis non cadit."[2] It will, I imagine, scarcely be contended that Celsus and the writer of the Hippocratic treatise are speaking of exactly opposite diseases, night-*blindness* and night-*sight* respectively. Exemptions so peculiar can only apply to similar diseases. The freedom from night-blindness of women in whom the catamenia are regular is mentioned by many other ancient Greek and Latin authors, though this alleged immunity has not been confirmed by modern authors, except that women are less exposed to the conditions which tend to induce night-blindness. In an epidemic of night-blindness which occurred in the district of Mausanne, it was noted by Fleury and Frechier[3] that *pregnant* women were most affected with it, though no age or sex was spared.[4]

*The appearance of eruptions about the ears*,[5] and *the supervention of abscesses and diarrhœa*,[6] are regarded as favourable prognostics in the Hippocratic nyctalopia. Though Bampfield, to whom we are indebted for the most trustworthy modern account of night-blindness, states that he could not perceive "that any particular constitutions or ages, or eyes of any particular description, form, or colour," were particularly subject to night-blindness;[7] he did find "a

[1] Prædict. ii, 40; Vander Linden, vol. i, p. 517; or, Prorrhet. ii, 33, 34, in Littré, t. ix, p. 64; or Prædict. II, in Foësius, sec. ii, p. 91. See also Adams's translation of "The Genuine Works of Hippocrates;" Syden. Soc.: London, 1849; vol i, p. 267.

[2] "De Medicina," lib. vi, c. vi.

[3] Quoted from the "Bulletin de Therapeutique," in Johnson's "Medico-Chirurgical Review," July 1842, p. 193; and cited by Mackenzie, *op. cit.*, p. 993.

[4] See also Dr. Grant's article, "Nyctalopia," in "Cyclop. of Prat. Med.," vol. iii, pp. 184, 185.

[5] "Epid.," iv.

[6] Prorrhet., ii.

[7] *Op. cit.*, p. 41.

spontaneous cure has sometimes succeeded to the eruption of boils on the face or head, and to the formation of abscesses of the face, head, or ears."[1] He also asserts that in cases of night-blindness associated with scurvy, "after a fresh animal and vegetable food has been instituted and persevered in for a short time, and the scorbutic diathesis is corrected, if a lax state of the bowels is not induced by the change of diet, as is usually the case, I have frequently directed cathartics;" adding that, though he had never administered cathartics without employing local treatment, he had, in several cases, observed so prompt a recovery from the combination, "that I have been induced to ascribe much of the benefit obtained to their use."[2]

*The Treatment.*—Perhaps the strongest argument that can be advanced in support of the opinion that the nyctalopia of Hippocrates, and that of the later Greek and Latin authors are the same, is the remarkable similarity in the reputed remedies. This is especially noticeable with regard to the local application of the bile or the sanies of liver, and to the ingestion of raw or roasted liver. The writer of the Hippocratic treatise "De Visu" recommends an ox's liver, but other writers seem to prefer that of the goat or the sheep. Galen, Celsus, and Pliny also mention and recommend this mode of treatment; indeed Pliny enters upon a long and detailed explanation of the *modus operandi* of goat's liver in curing persons who cannot see by night (*nyctalopas* a Græcis dictos).[3] Goat's or sheep's liver, or the liver of some other animal, is also recommended by Oribasius,[4] Marcellus,[5] Aëtius,[6] Alexander Trallianus,[7] Paulus Aegineta,[8] and Actuarius.[9]

[1] *Op. cit.*, p. 49.

[2] *Op. cit.*, p. 51.

[3] "Hist. Nat.," lib. xxviii, c. 47. *See*, further, Dr. Greenhill's letter, p. 285, notes 4 and 5.

[4] "De loc. Affect. cur.;" lib. iv, c. 18.

[5] "De Medicamentis," cap. viii.

[6] Tetr. ii, serm. iii, c. xlvi.

[7] "De Arte Med.," lib. ii, c. vi.

[8] Lib. iii, c. xxii.

[9] "Med. sive Meth. Med.," lib. iv, c. xi.

Guillemeau[1] refers to it, and Scarpa,[2] citing Dupont,[3] also mentions it. Adams, in his commentary on Paulus Aegineta,[4] states that Rhases, Avicennna, Mesue, Jesu Haly, Haly Abbas, and Alsaharavius also speak favourably of the application of the roasted liver in cases of night-blindness.

Even in recent times the hepatic treatment of night-blindness has been employed. Dr. Grant[5] quotes the experience of Dr. Meissener, and states that he himself "has repeatedly seen a cure *apparently* produced by fumigating the eyes with the vapour of a bullock's liver," though he does not believe that there is any specific virtue in the liver.

*The concomitants* of Hippocratic nyctalopia are swelling of the gums, toothache, foul breath, swellings below the eyes, bleeding from the nose, enlarged spleen, and ulcers on the legs.[6] I need scarcely say that this train of symptoms indicates the condition now called Scorbutus, of which night-*blindness* is a well-known symptom. The account given by Hippocrates may not be a perfectly accurate description of scorbutus in all its parts, but it is sufficiently correct to enable us to identify it. Van Swieten pertinently remarks: "Although it cannot be denied but the ancient physicians have reckoned up many of the symptoms of the scurvy in their descriptions of several distempers, handed down to us under a different denomination; yet it is not so apparent that they have seen into the real nature of the distemper itself, nor so plainly described the symptoms of it, that one may be able from them to gather a full knowledge of the scurvy."[7] Of the accounts given in the Hippocratic writings, he remarks, "though he [Hippocrates] mentions a great many symptoms of the scurvy, seems not to have judged that

[1] *Op. cit.*, pp. 27, 28.

[2] *Op. cit.*, p. 254; and vol. ii, p. 323, of the French translation.

[3] "Mémoire sur la goutte sereine nocturne épidémique, ou Nyctalopie."

[4] Book iii, sec. 22; Syd. Soc. ed.; vol. i, p. 434.

[5] *Op. cit.*

[6] "Prorrhet.," ii.

[7] "Comment in Hermanni Boerhaave Aphorism:" Lug. Bat., 1753, t. iii, p. 590; or Eng. trans.: Lond., 1754, vol. xi, p. 278.

they all concurred together to make up one particular distemper, but supposed them to come from some defects in spleen."[1]

It would lead me too far to trace out all the points of resemblance, nor is it necessary for the purpose of this article that I should do so. I would, however, call attention to the fact that all modern observers are agreed that night-blindness is a frequent concomitant of scorbutus. Wolfgang Hoefer states that in the time of the Swiss war he had seen many soldiers and others suffering from night-blindness, due to the scarcity of provisions. These were all cured by an improved diet (curantur verò tales mutatione diætæ in meliorem).[2] Sir Gilbert Blane states that night-blindness, as a symptom of scurvy, had been reported to him by Mr. Telford,[3] and Dr. W. Kerr alleges that it was a common occurrence among the scorbutic patients in the garrison of Gibraltar, during the siege of that place.[4] Andrew Simpson also mentions the frequent co-existence of night-blindness and scurvy.[5] The association has, however, in recent times been especially insisted on by Bampfield,[6] and has been confirmed by others. In some cases of scurvy occurring in H.M. brigantine "Griffon," and recorded by Dr. Bryson, night-blindness was the first symptom of the disease.[7]

The frequent association of scorbutus and night-blindness will not be disputed, and I have little hesitation in expressing the opinion that the many of the "nyctalopes" of Hippocrates were actually suffering from scurvy. If this opinion be well-founded, it lends powerful and independent support to the amended reading of *οὐχ ὁρῶντες*, in "Prorrhet.," ii, inasmuch as night-*sight* has never yet been observed as a concomitant of scorbutus, whereas night-*blindness* is often present.

[1] *Op. cit.*, t. iii, p. 591; or Eng. trans., xi, p. 281.

[2] "Hercules Medicus:" Noriberg, 1675; lib. i, c. viii, p. 72.

[3] "Observations on the Diseases of Seamen," 3rd ed.: Lond., 1799.

[4] Art. "Scorbutus," in Cyclop. of Pract. Med.: Lond., 1834; vol. iii, p. 684.

[5] *Op. cit.*

[6] *Op. cit.*, p. 33, *et seq.*

[7] "Ophth. Hosp. Rep.," vol. ii, p. 40; July, 1859.

This hypothesis likewise furnishes a clue to the *rationale* of the alleged cure of nyctalopia by means of bullock's or goat's liver, as related above. The fresh roasted liver, whether of a bullock, goat, sheep, black-cock, or other animal would act beneficially on the scorbutic condition of the body, and would in this way expedite the cure of the disordered vision. It is worthy of note, too, that many of the other remedies recommended in the Hippocratic collection for the cure of nyctalopia are also serviceable in scurvy, and that the favourable prognostic signs are also those of scorbutus.

(5). For the fuller consideration of the fifth reason, namely, that there is good manuscript authority for the belief that the reading in the second book of the Prorrhetics should be that persons who do *not* see at night are called nyctalopes, I must, beyond what I have already incidentally remarked, refer the reader to Dr. Greenhill's letter and criticism, and especially to the valuable note on page 287. I may, however, observe that an omission of *οὐ* or *οὐκ* from ancient manuscripts is by no means rare. Such an omission occurs in the Hippocratic treatise *De Internis Affectionibus*, where persons who have ulcers on the legs with the skin black and thin, are said to be eager to walk about and to make exertion.[1] This should obviously be read, "are *not* disposed to walk about or to make exertion," and is so read by Littré[2] and Vander Linden,[3] both of whom supply the *οὐ*. The omission of the word *οὐ* is, in this instance, as in the second book of the Prorrhetics, obviously a fault of the copyist. The text in the latter instance, as Chamseru, Coray, Ermerins, and Dr. Greenhill believe, should be *οἱ δὲ τῆς νυκτὸς ΟΥ'Χ ὁρῶντες. κ.τ.λ.* Coray remarks[4] that the inad-

[1] Foësius, "Hippoc.," sec. v, p. 119; Linden, ii, 256.

[2] *Op. cit.*, t. vii, p. 280.

[3] *Op. cit.*, vol. ii, p. 256. It is an odd circumstance that while Vander Linden inserted *οὐ* in the Greek text of his edition of Hippocrates, he did not correct the Latin translation, which is, "Est autem ad deambulandum ac laborandum promptus."

[4] "Traité d'Hippocrate des Airs, des Eaux, et des Lieux:" Paris, 1800; vol. ii, p. 45.

vertent omission of *οὐ* is especially common after words ending in *ος*, as in this instance (*νυκτὸς*), or in the treatise "De Aëribus, Aquis, Locis," where he has inserted *οὐ* between *πλῆθος* and *γλυκαίνεται*.[1]

The adoption of the reading *οὐχ ὁρῶντες* would not only get rid of the otherwise inexplicable confusion, but would furnish a meaning of the term nyctalopia more in accordance with the principles of etymology than *night-sight* supplies. Indeed, unless we are prepared to accept Voltaire's satirical definition of etymology, as "une science où les voyelles ne font rien, et les consonnes fort peu de chose," we must, independently of any other testimony, recognize the negative or privative element in the words "nyctalopia" and "hemeralopia." To employ the word nyctalopia as night-sight, and hemeralopia as night-blindness, seems, as I have already stated (p. 417), a philological extravagance and absurdity.[2]

The contention that true *night-blindness* was not known in the time of Hippocrates, has been already met by anticipation in the comments on Aristotle's explanation of the differences of colour in the eyes, and of the causes of night-blindness in persons who have *dark* eyes. (*See* p. 429.)

On these grounds we must, I think, agree with Galen, that the true meaning of nyctalopia is night-blindness (*ὁ τῆς νυκτὸς ἀλαός*), and, consequently, that the true meaning of hemeralopia is day-blindness.

Unless, therefore, we are disposed to adopt the suggestion of the late Dr. Mackenzie, not to use these terms at all, in any sense, we should retrace our steps, and abandon the later (false) meanings for the original and true ones.

[1] "Traité d'Hippocrate des Airs, des Eaux, et des Lieux:" Paris, 1800; vol. i, p. 14. Littré (t. ii, p. 18) gets over the difficulty by substituting the word *ἐγγίγεται* for *γλυκαίνεται*; while Adams, in his translation (vol. i, p. 193), omits the clause altogether.

[2] See further, Dr. Greenhill's critical remarks, p. 288, *et seq.*

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